Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

Kaapstad 20 Februarie 1960 Deel 34 No. 8 Volume 34 Cape Town, 20 February 1960

General Practice Series

ANAESTHESIA FOR MINOR GYNAECOLOGICAL OPERATIONS

C. S. JONES, M.B., CH.B., *Head of the Department of Anaesthesia, University of Cape Town and Groote Schuur Hospital*

In speaking of minor gynaecological operations I am referring particularly to evacuations of the uterus for incomplete abortion, diagnostic dilatation and curettage, hysterograms, the insertion of radium for the treatment of carcinoma, cauterization of the cervix uteri, drainage of Bartholin's abscess, and the many other minor procedures which aid in the diagnosis and treatment of the lesser ills of the female.

These minor procedures make up a formidable part of the total volume of operative gynaecology. At Groote Schuur Hospital in 1958 a total of 2,768 anaesthetics were administered for the commoner minor procedures in gynaecology. The total of gynaecological anaesthetics was 4,194 so that these particular minor procedures made up 66%. They included the following:

Evacuations for incomplete abortion	1,192
Diagnostic dilatation and curettage	749
Diagnostic biopsy and cauterization	429
Hysterograms	192
Insertion of radium	206
Total	2,768

These operations seldom require prolonged anaesthesia, nor do they call for deep anaesthesia. Many of the patients do not need to be hospitalized at all, or else they need to be kept in hospital overnight only.

In general the patients are reasonably fit, with the obvious reservation that some of them, especially amongst the group requiring evacuation for incomplete abortion, and particularly those who require this treatment urgently to stop haemorrhage, may well be gravely ill from acute blood loss, and must be handled with despatch but with more than ordinary caution. But the great majority do not present any real problems from the point of view of the anaesthetist and are, as a rule, quite safely and speedily anaesthetized along the lines I wish to describe.

AIMS OF ANAESTHESIA

It is too often forgotten that the anaesthetic technique should be tailored to suit the requirements of the type of surgery to which the patient is to be subjected. As an extreme example of 'frozen foresight' I cite the example of the patient who was given, by a specialist anaesthetist, an anaesthetic admirably calculated to provide suitable facilities for cholecystectomy, viz. endotracheal intubation under thiopentone and a relaxant, but one far too elaborate and hazardous for the diagnostic curettage for which the patient was scheduled and to which she was actually subjected. This was an example of sending a man on a boy's errand. When I see such displays I am moved to speculate, uncharitably, that the choice of anaesthetic is perhaps dictated, not by the needs of the patient, of the surgeon or of the surgery, but those of the anaesthetist's ego; and 'egotistical anaesthesia' is perhaps not undescriptive of the rut into which that anaesthetist has sunk.

Needs of the Patient

The lady who is to have one of the minor operations we are discussing will almost certainly be placed in the lithotomy position as being the one best calculated to assist the surgeon. It is, however, an undignified position and not one the patient would care to assume while conscious and in the presence of onlookers. From that point of view unconsciousness is something she would appreciate. She usually has no other demands to put to the anaesthetist except the unvoiced, but assumed, wish to be spared any pain while awake or asleep.

Needs of the Surgeon

The surgeon who is to perform the operation (and at Groote Schuur Hospital 76% of the gynaecological surgery in 1958 was done by registrars and interns) wishes to have a patient with relaxed abdominal wall so that he may palpate her pelvic organs with ease. The lithotomy position flexes the thighs on the trunk and flexes the trunk also, so that the abdominal muscles are well relaxed in this position, and the anaesthetist needs only to prevent any active muscular contraction to satisfy the surgeon on this score. Adequate anaesthesia with the avoidance of any hypoxia and hypercarbia will provide this quiet musculature. Drug-induced muscular paralysis, either from great depth of anaesthesia or from the use of curare, is unnecessary and, indeed, to be condemned for such brief operations as these. Any other demands the surgeon may make are usually unwarranted and run parallel, too often, with the old adage that it is only the inept workman who blames his tools or his assistant for a job badly done.

The cervix cannot be relaxed by deep anaesthesia or by curare.

Needs of the Anaesthetist

The anaesthetist asks that his anaesthetic shall act with rapidity, produce relief from any pain or discomfort of the operation, and then release the patient to a rapid return to consciousness and a minimal degree of after-effects such as nausea, headache or spots-before-the-eyes.

In selecting his agents and techniques he must bear in mind the needs of the patient and the surgeon which have just been discussed. He must remember, too, the needs of the surgery and the anatomy and physiology of the parts

which are to be subjected to surgical interference. These parts were designed to absorb a considerable amount of physical punishment, and have been innervated accordingly, chiefly through the autonomic nervous system. Their threshold of perception, moreover, varies with the psychical state of the patient.

Under conditions requiring digital examination of the pelvic organs, touch is not well appreciated unless it causes pain, and pain is usually only produced if there is local inflammatory reaction or unusual trauma on the part of the examiner. Thus, for the vast majority of patients undergoing minor gynaecological surgery, examination *per vaginam*, which is always an important part of any procedure, may be uncomfortable but is not likely to be painful, so that no great depth of anaesthesia will be required to prevent reflex increase in tone of the abdominal muscles. The lithotomy position, except in the aged arthritic, is not painful, nor is catheterization of the female bladder—a usual prelude to operations in this area. The single manœuvre which is most likely to cause pain is the rapid, forceful dilatation of the cervix uteri.

The cervix can be dilated in a conscious, unanaesthetized patient without pain *only* if the dilatation is done very gently, very slowly, and with an almost infinite gradation in dilator sizes. As far as is known (and anatomy textbooks read like 17th century treatises on this aspect) the cervix is not innervated from the central nervous system at all but only from the autonomic system, so that *tension* is the only pain-stimulus the cervix will appreciate. Thus, the cervix can be seized, crushed, cut or burned without pain, as is well known by professional abortionists.

There lingers still, in reputable anaesthetic text-books, the century-old observation that rapid, forceful dilatation of the anal sphincter or the cervix causes pain such that only deep anaesthesia can obtund and that, as a result, these manœuvres can be resorted to as a last measure in resuscitating the patient apparently dead from an anaesthetic. It is of interest to uncover the origin of this myth, which dates back to John Snow, the first specialist anaesthetist, and James Young Simpson who introduced chloroform to gynaecology. In those days patients were exhorted to 'breathe deeply' as anaesthesia was being induced and they usually obliged. In addition to aiding in the uptake of the anaesthetic vapour, this overventilation washed out carbon dioxide. As a result, and particularly in susceptible patients, the blood pressure fell [1-3] and the patients became apnoeic although only very lightly anaesthetized. Under these circumstances of apparently approaching death, *any* painful stimulus will cause the patient to gasp. It needed only a single observation of this phenomenen to brand the value of this 'resuscitative measure' on the brain of every anaesthetist of that generation and, indeed, of every succeeding generation. How many other empirical measures in the practice of medicine are based on as tenuous a coincidence?

The method of providing anaesthesia for minor gynaecological operations which I am about to describe takes into account all that I have outlined above, and can be speedily and *safely* applied to at least 80% of patients presenting for such surgery. The remaining 20% can be handled as safely with intelligent modifications of the technique, but in this respect I draw particular attention to the last, but by no means the least, of the requirements listed below.

A METHOD OF ANAESTHESIA FOR MINOR GYNAECOLOGICAL OPERATIONS

Required

1. A patient who has been properly examined and prepared for an anaesthetic, including the pre-anaesthetic administration of hyoscine hydrobromide, 0·4 - 0·6 mg. by injection, with or without morphine sulphate, 6 - 10 mg., or pethidine, 20 - 50 mg. Atropine (also in a dosage of 0·4 - 0·6 mg.) can be used in place of hyoscine but is less valuable.

2. An anaesthetic machine capable of delivering nitrous oxide and oxygen mixtures in the ratio of 8 l./min. of nitrous oxide and 2 l./min. of oxygen (20% of oxygen).

3. A 20 - ml. syringe fitted with a sharp, sterile needle of adequate size and charged with 20 ml. of a 2·5% solution of sterile thiopentone.

4. An intelligent and alert anaesthetist.

Method

The patient is placed on the operating table and an arm extended on an armboard. A sphygmomanometer cuff is fitted snugly to this arm above the elbow and the patient's blood-pressure is measured and recorded. The cuff is then re-inflated to a pressure midway between systolic and diastolic pressures and a suitable vein sought.

When the vein has been found and is maximally distended with blood (the cephalic or basilic veins just below the elbow are the best), the needle of the thiopentone syringe is inserted into it, the cuff deflated and 2 ml. of the thiopentone injected. The syringe is fixed to the arm with adhesive tape, the anaesthetist moves to the patient's head, and while he is turning on his nitrous oxide *and* oxygen in the ratios described, he asks the patient, 'do you have any pain in your hand?' The answer to this question tells him (*a*) whether he is injecting into an artery, and (*b*) how much thiopentone the patient is likely to need. The patient who is already asleep from 50 mg. of thiopentone is not likely to require very much more.

When he has turned on his gases and has obtained the answer to his question the anaesthetist injects a further 4 - 6 ml. of thiopentone, and announces pleasantly, 'I am going to give you some gas as well, while you are going to sleep', and gently applies the mask of the anaesthetic machine to the patient's face to commence the administration of nitrous oxide and oxygen, the chief prop of his anaesthetic technique.

He then announces, 'Mrs. X, we are going to bend up your legs and wash you. Just relax and lie still'. Mrs. X may very well not hear this, but it is surprising how effective this psycho-anaesthetic approach is. At this stage the surgeon and nursing staff, with gentle, deliberate movements, procede to position the patient and clean the vulva and vagina with antiseptic solution. Seldom is additional thiopentone needed for this but it can, of course, be given if needed.

If the surgeon is a person who uses tinctures on the vaginal mucosa, he is best avoided. Alternatively he can be prescribed a Sitz bath in alcohol for himself.

The draping of the patient follows the cleaning and is a painless procedure, unless towel clips are unnecessarily inserted into the patient's skin. Painless also is the catheterization of the bladder, but it is a signal to the anaesthetist that the examination of the pelvic organs is shortly to begin.

He therefore, in anticipation, administers another dose of 4 - 6 ml. of thiopentone. As he had 10 - 14 ml. in his syringe before this, he will have at least 4 ml., and perhaps as much as 10 ml., in reserve for the balance of the operation, after giving this pre-examination dose, which should be so timed that administration of the dose ceases at least 45 seconds before the surgeon inserts his hand into the vagina.

When the examination has been completed, the operation begins. Even if the cervix is to be dilated, no further thiopentone is administered until the patient stirs, when another dose of 2 - 4 ml. is usually ample to quiet her.

The anaesthetic is continued in this way, with intermittent doses of thiopentone supplementing a basic nitrous oxide and oxygen anaesthetic, until the operation is ended. It is very seldom necessary to use more than a total of 500 mg. of thiopentone. Ill and anaemic patients will need less, while those few patients who will obviously need more are introduced early in the course of the anaesthetic to a more potent supplement, such as trilene or ether.

The blood pressure is determined from time to time and a remarkable volume of information on the behaviour of the cardiovascular system under anaesthesia can be acquired in this way.

When the operation has been concluded the syringe and needle are removed from the arm and the mask from the patient's face. Most patients will be waking up as they leave the operating theatre and very few will be still asleep when they reach their bed.

Some Don'ts

(*a*) Don't jerk any part of the patient suddenly and roughly. It may cause coughing and laryngeal spasm.

(*b*) Don't use any harness to hold the anaesthetic mask in place. The anaesthetist's hand is best for this purpose and the use of airways is avoided. The mask can be removed more quickly also, in an emergency.

(*c*) Don't use any airway if it can reasonably be avoided. These patients are usually so lightly anaesthetized that they will cough or vomit if an airway is used, and may develop laryngeal spasm.

(*d*) Don't exceed a total dose of 500 mg. of thiopentone except under completely justifiable circumstances.

(*e*) Don't give the patient less than 20% of oxygen or a total gas-flow volume of less than 10 litres per minute. To do this will lead to *anoxia* and *hypercarbia*. Anoxia causes restlessness and fighting during anaesthesia and nausea and vomiting during recovery. Hypercarbia causes muscular contraction during anaesthesia and leads to apnoea and a fall in blood pressure during recovery.

(*f*) Don't allow your attention to wander *in any way* from the patient, the anaesthetic she is receiving, and the operation she is being subjected to. Accidents always seem to happen when one is not looking.

The figures from Groote Schuur Hospital are quoted with the permission of the Medical Superintendent, Dr. J. G. Burger.

REFERENCES

1. Henderson, Y. (1908): Amer. J. Physiol., **21**, 126.
2. *Idem* (1909): *Ibid.*, **25**, 310.
3. Hill, L. and Flack, M. (1910): J. Physiol., **40**, 346.

DIABETIC EMBRYOPATHY*

W. P. U. JACKSON, M.D., M.R.C.P., *Department of Medicine, Groote Schuur Hospital and University of Cape Town*

The diabetic mother frequently gives birth to a foetus with several peculiarities. A high proportion of her infants are over-weight, often over 10 lb., and this increase in weight is made up of several components. The babies are both over-long and over-fat; they are oedematous and characteristically lose a pound or so of fluid weight during the first few days of life. Their internal organs are large; in particular there is cardio- and hepato-megaly. They may have an expanded erythron, with widespread haematopoiesis and a high haemoglobin content in their blood. Their general appearance of fat, flabby, rubicund weakness may closely resemble the picture of Cushing's syndrome.

These babies, and also those which escape being overlarge, behave as if they were feeble, undersized, premature infants; they have difficulty in breathing and sucking, and easily regurgitate fluids into their lungs. They are susceptible to birth trauma, infection, and hyaline membrane disease, so that they may die within the first few days of life unless specially protected. Although their blood-sugar levels may be very low soon after birth, this is probably of no significance, since a normal baby may likewise have extreme hypoglycaemia without any symptoms from it.

Foetuses of diabetic mothers probably show an increased incidence of major congenital anomalies, some of which, such as anencephaly and ectopia cordis, may be incompatible with life. Quite apart from such anomalies, of course, a number of foetuses are not born alive. They die about the 36th week or earlier and are delivered in a macerated state, or they die nearer term and are delivered in a better state of preservation. Although these stillbirths are near or even at full term, they are distinguished by active hepatic haematopoeisis, similar to that seen in premature and erythroblastotic infants. Luteal cysts may be found in the ovaries. Warren and Le Compte,[1] and Cardell,[2] in particular, have described the pathological features of these infants in more detail, but I think the most interesting abnormalities reside in the pancreas. Almost invariably the islets of Langerhans in these pancreases are large, and occupy an excessive part of the whole organ. Both cellular hyperplasia and cellular hypertrophy are present. Further than this, there is an increased proportion of beta cells in the islets. In the normal newborn infant the alpha cells comprise 60 - 70% of islet tissue—the figure is reversed in the infant of the diabetic mother. The sections we have examined appear, moreover, to show an unusual degree of granularity in the beta cells which, following Wrenshall and Hartroft,[3] may indicate an excess of insulin content. In fact, if we take all these items together it looks as if the pancreas of the diabetic's infant may contain up to 30 times the normal amount of insulin.

This abnormal condition of the embryo, or 'embryopathy', as it has rather unfortunately been called, owes its existence to some abnormality in the diabetic mother, since it is not seen in the infants of diabetic fathers. Certainly we have found,[4] and it has been confirmed by Pirart,[5]

* Part of a contribution to symposium on 'Diabetes and Pregnancy', in the 3rd Congress of the International Diabetes Federation, Düsseldorf, West Germany, 21-25 July 1958.

that the birth weights of the infants of diabetic fathers are larger on the whole than those of non-diabetics, but this increase in birth weight is not nearly so striking as in the infants of diabetic mothers, and there is no increase whatever in the stillbirth rate in the progeny of diabetic fathers.

Now what types of diabetic are liable to such an embryopathy? Here we come across some discrepancies. In most centres of the world the same type of baby has been found to be produced by the severe, insulin-requiring, growth-onset diabetic, by the mild, diet-controlled diabetic, and even by the prediabetic several years before she shows any obvious metabolic carbohydrate disorder. In Boston[6] and Brussels,[5] apparently, the tendency for foetal loss to occur in the mild diabetic and in the prediabetic is very much less, and the data from these cities seem to be unimpeachable. In Cape Town we found evidence that the tendency to produce stillbirths and large babies extends back indefinitely into the past obstetric life of the diabetic woman, in her prediabetic phase.

In fact, in our experience all the above-described features of the diabetic's infant apply with equal force to the infant of the prediabetic. It was Van Beek[7] who first emphasized that the enlarged islets of Langerhans were to be found in the stillbirths of women who only later became diabetic. We have amply confirmed this.[8] The mean percentages of islet tissue in stillborn pancreases in control stillbirths was 1·3% by our method, while it was around 7% in erythroblastotics, in children of diabetic mothers and in children of prediabetic mothers. It is extraordinary that once again this similarity is seen between erythroblastosis and diabetes. We know of no other cause for such enlarged islets in the stillborn infant. In fact, the finding of enlarged islets in a stillborn which is not erythroblastotic and whose mother is not an overt diabetic, is the best possible indication of prediabetes in the mother. Our results illustrate this: Pancreases from 109 autopsies on stillbirths were specially examined and their islet contents estimated; 18 were found in which the proportion of islet tissue was over 5·5%. Of the 18 relevant mothers, 12 were traced for follow-up; 5 were found to have become diabetic, 5 gave slightly abnormal glucose tolerance curves, of whom 2 are now plainly diabetic, and in the other 2 there was strong collateral genetic and obstetrical evidence of prediabetes. Thus we believe all 12 mothers to have been prediabetic when they gave rise to the stillborn infants with large islets of Langerhans.

Looked at from another angle, these findings may provide a pointer in hitherto unexplained stillbirths, since the finding of large islets will indicate that maternal diabetes or prediabetes has played a part in the foetal death.[9]

These excursions into the realms of prediabetes may help us in our search for the cause of this strange embryopathy. It is certainly most unlikely that maternal hyperglycaemia can play any part, since many prediabetics show normal blood-sugar levels during pregnancy, and yet produce abnormal infants.[10] A sensitivity to, or excessive production of, growth hormone or glucocorticoids in the diabetic pregnancy has been suggested and, although Professor Hoet[11] has made some interesting experiments the results of which favour the participation of glucocorticoids in this syndrome, the evidence is as yet not very good in favour of either. Although the plasma-cortisol level rises during all pregnancies, the levels observed are no higher in the diabetics. We have not found any tendency for women who are in the early stages of acromegaly or Cushing's syndrome to produce large babies or stillbirths, and this is surely strong evidence against either growth hormone or corticoids being the sole cause of the embryopathy.[10] The large pancreatic islets suggest an excessive stimulation of this tissue. Is the infant's own insulin, acting as a 'growth hormone', itself in fact the stimulus to the excessive size? And what is the connection with the large islets in erythroblastosis?

Turning finally to the prevention and management of the embryopathy, Professor Hoet[12] and Dr. Wilkerson[13] believe that insulin given to the prediabetic during pregnancy, even when her blood sugar is normal, may be effective in preventing some of the features of the embryopathy. And yet in the established diabetic, the very best possible control of the mother's diabetes will reduce the incidence of abnormal babies only partially. To obtain a live child it appears to be even more important to induce labour early if the foetus is judged to be large enough, and to manage the baby exactly as though it were truly a premature infant. Before this can be done, of course, it is necessary to make a diagnosis of maternal diabetes or prediabetes, and I am sure that a number of babies may be saved if the latter diagnosis is suspected and the suspicion is acted upon.

I should like to thank Messrs. Hoechst Pharmaceuticals of South Africa and the parent company in West Germany, and also Noristan Laboratories, of Pretoria, and Messrs. C. H. Boehringer Sohn, of West Germany, for their great assistance in making possible my attendance at this Congress.

REFERENCES

1. Warren, S. and Le Compte, P. M. (1952): *The Pathology of Diabetes Mellitus*, 3rd ed. Philadelphia: Lea and Febiger.
2. Cardell, B. S. (1953): J. Path. Bact., **66**, 335.
3. Hartroft, W. S. and Wrenshall, G. A. (1955): Diabetes, **4**, 1.
4. Jackson, W. P. U. (1954): J. Clin. Endocr., **14**, 177.
5. Pirart, J. (1955): Acta. endocr., **20**, 192.
6. Wilkerson, H. L. G. and Remein, Q. R. (1957): Diabetes, **6**, 324.
7. Van Beek, C. (1952): *Autopsy findings in stillbirths and neonatal deaths suggesting maternal diabetes*. Presented at the First International Congress of the International Diabetes Federation, Leyden, The Netherlands, 7-12 July.
8. Woolf, N. and Jackson, W. P. U. (1957): J. Path. Bact., **74**, 223.
9. Jackson, W. P. U. and Woolf, N. (1958): Diabetes, **7**, 446.
10. Jackson, W. P. U. (1955): Lancet, **2**, 625.
11. Hoet, J. P. (1954): Diabetes, **3**, 1.
12. Hoet, J. J., Gommers, A. and Hoet, J. P. (1958): 3rd Congress of International Diabetes Federation, Düsseldorf, 24 July.
13. Wilkerson, H. L. G. (1958): Personal communication.

VISIT BY DR. J. ALLISON

Dr. J. Allison, Director of the Bureau of Biological Research, Rutger's University, New Brunswick, New Jersey, USA. will be in Cape Town on 26 and 27 February. He is an international expert on protein requirements and a leading research worker in experimental protein deficiency in dogs. Dr. Allison will give a brief talk during the lunch hour on Friday 26 February in the Conference Room of the Department of Medicine, University of Cape Town, Observatory, Cape, at 1.15 p.m. Tea will be provided at 1 p.m. for those who wish to bring sandwiches.

VAN DIE REDAKSIE : EDITORIAL

ONDERBREEKTE POSITIEWE-DRUK ASEMHALING: FISIOLOGIESE BEGRIPPE

Daar is niks nuuts in kunsmatige asemhaling deur middel van direkte, onderbreekte verhoging van die lugdruk in die tragea nie.[1] Die gebruik van onderbreekte positiewe-druk asemhaling (O.P.A.) in die algemene medisyne oor lang tydperke is egter slegs ingestel sedert die tegniese vooruitgang wat teweeggebring is deur die werk van Lassen gedurende die Deense kinderverlammingepidemie van 1953. Lassen en die narkotiseur Ibsen het beweer dat die pasiënt met verlamming van die sluk- sowel as borsspiere, baie na aan die pasiënt staan wat verlammingsmiddels vir chirurgiese doeleindes ontvang het.[2] Derhalwe het hulle die narkotiseur se tegniek geleen: lug word ritmies uit 'n sak in die longe gedruk deur 'n lugdigte endotrageale buis. Weens die verwoesting van die stembande wat die endotrageale buis binne 'n paar uur teweegbring, is 'n trageostoom gebruik om 'n dergelike maar korter buis in die tragea te plaas. Hoe skofte van mediese studente die lug ingestoot het, is 'n welbekende mediese verhaal. Die volgende stap was om 'n ‚meganiese student' te gebruik, en vandag is daar 'n groot verskeidenheid van O.P.A.-pompe verkrygbaar.

Alhoewel kinderverlamming minder dikwels voorkom, neem die gebruik van O.P.A. steeds toe. Benewens die toepassing in ander verlammende siektes soos polineuritis en miastenie gravis, word dit gebruik in uitgesoekte gevalle van emfiseem, klemkaak, slaapmiddelvergifting, kopbeserings, en die versteurde asemhaling van die vettes.

In Noord-Amerika is dit gebruiklik om die liggaamsmasjien (ysterlong) te gebruik al het die pasiënt 'n trageotomie nodig, maar die beginsels verskil geensins van O.P.A. nie.[3] In albei word die trageale druk verhoog bo die druk op die liggaamsoppervlak. O.P.A. doen dit direk deur die druk in die windpyp te verhoog; die ysterlong verminder die druk op die liggaamsoppervlak. Albei maniere van asemhaling verhoog die borskasdruk bo die druk op die periferele are, en derhalwe word die veneuse terugvloei na die hart in albei gevalle verminder.[4]

Dit is derhalwe heeltemaal onjuis om te beweer dat die ysterlong ‚meer fisiologies' is. Die mate van hierdie verhindering word die maklikste gemeet deur die slukdermdruk, wat 'n redelike indeks van die druk in die mediastinum is en derhalwe ook van die druk wat op die regterhart toegepas word. Deur middel van 'n vergelyking van die uitwerking van normale en kunsmatige asemhaling op die slukdermdruk is dit moontlik om 'n meer juiste idee te verkry van die uitwerking op die veneuse terugvloei.

In die verlamde pasiënt wat onderbreekte positiewe-druk asemhaling van 'n standaard pomp soos die Radcliffe-model ontvang, is die gemiddelde borskasdruk gewoonlik tussen 3·5 en 4 sm. water hoër as in die gesonde mens.[5] Die gesonde hart is heeltemaal daartoe in staat om hierdie obstruksie te oorkom, maar as die pasiënt kunsmatige asemhaling nodig het, is daar dikwels ook 'n versteuring van die bloedsomloop, veral in siektes soos kinderverlamming en polineuritis. Dan word dit van groot belang om die veneuse terugvloei aan te help; die gebruik van 'n negatiewe-lugdrukfase gedurende die uitasemingsperiode is die maklikste manier om hierin te slaag. Wanneer negatiewe druk gebruik word, val die gemiddelde borskasdruk na aan die waarde van dié van die normale mens. Die wissellug (tidal volume) word ook vergroot.

Die lengte van die inasemingsperiode moet ook streng ingekort word, veral as die sogenaamde ‚vierkantige' positiewe lugdrukpatroon gebruik word, omdat daar eenvoudig nie meer lug in die longe ingaan na 1·0 tot 1·5 sekonde se inaseming nie; terselfdertyd word die veneuse terugvloei onnodiglik verhinder.[5] Die uitasemingsperiode moet ook minstens twee derdes van die asemhalingsiklus behels, en verkieslik moet daar dan ook 'n negatiewe druk wees.[6,7]

Onderbreekte positiewe-druk asemhaling het 'n bepaalde plek in die behandeling van die pasiënt met gevorderde emfiseem en kooldioksied-retensie. Die gebruik van 'n negatiewe druk is egter geen waarborg dat die borskasdruk beïnvloed gaan word nie; inderdaad, daar is regtig 'n gevaar dat die negatiewe druk die bronchi sal toesuig, met die gevolg dat lug in die longe gevang word met die verhoging van die borsdruk en skadelike gevolge op die veneuse terugvloei na die regterhart.[8] Die gevolg is onvermydelik: die positiewe fase stoot die borsdruk op en die negatiewe druk neig om dieselfde te doen. Daarom sou dit raadsaam wees om versigtig te wees met die gebruik van onderbreekte positiewe druk in pasiënte met sowel emfiseem as 'n neiging tot hartversaking. Gevalle met uitgesproke regterhartversaking is egter suksesvol behandel deur O.P.A.[9]

Onder gewone omstandighede is die bloedsomloop egter daartoe in staat om bevredigend te reageer op die drukverandering gedurende meganiese kunsmatige asemhaling, tensy die positiewe fase onnodig verleng word of die bloedsomloop direk aangetas is. In sulke gevalle moet elke moontlike middel gebruik word om die borskasdruk te verlaag—tensy daar ook emfiseem is, wanneer 'n negatiewe druk moontlik meer skade as goed kan doen.

1. Hook, M. (1667): Phil. Trans. B., **2**, 539.
2. Lassen, H. C. A. (1953): Lancet, **1**, 37.
3. Maloney, J. V. en Whittenberger, J. L. (1951): Amer. J. Med. Sci., **221**, 425.
4. Spalding, J. M. K. (1959): Proc. Roy. Soc. Med., **52**, 664.
5. Opie, L. H., Spalding, J. M. K. en Stott, F. (1959): Lancet, **1**, 545.
6. Berneus, B. en Carlsten, A. (1955): Acta med. scand., **152**, 19.
7. Cournand, A., Motley, H. L., Werkö, L. en Richards, D. W. (1948): Amer. J. Physiol., **152**, 162.
8. Annotation (1959): Lancet, **2**, 60.
9. Barber, H. O., Chambers, R. A., Fairley, H. B. en Woolf, C. R. (1959): Canad. Med. Assoc. J., **81**, 97.

EMPTYING THE COLON

Quite a few members of the allegedly civilized section of the human race devote considerable thought and time to opening their bowels and to keeping them moving in spite of the fact that we have been so formed that visceral activities do not demand our constant supervision. The idea that there was great virtue in having a complete 'turn out' is now not generally held. The weekly dose of jalap-and-ginger or brimstone-and-treacle has fallen into disuse and is now as outmoded as Lane's colectomy.

There are, however, a few important diagnostic pro-

cedures which are greatly facilitated by a completely empty colon and the means to attain this desirable end are many and various. For example, the distal large bowel must be entirely clear before sigmoidoscopy can be performed. The whole colon must be empty before a barium enema can be carried out, and the absence of solid and gaseous large-bowel contents is appreciated by urologist and radiologist alike when it comes to the study of intravenous pyelogram films.

The preliminary IVP preparation is often omitted entirely or the patient is casually asked to take a dose of opening medicine the night before. The resulting partial obliteration of the renal anatomy allows the radiologist to sit on the fence and state that large-bowel contents obscure detail, but as far as he can see there is no gross abnormality present. This in turn irritates the urologist and he probably likewise cannot be certain that all is well; and so finally the patient is subjected to the expense and discomfort of retrograde pyelography before an opinion is finally reached. It would have been kinder to have been firm with the patient in the first place and given him something efficacious.

In the past an enema was given or a bowel wash-out done as a routine before sigmoidoscopy or barium enema and quite often used before an IVP. Most people, however, do not take kindly to the thought (and practice) of being impaled upon a rubber tube in strange surroundings and then being well and truly washed out. The most comfortable padded and chromiumed colonic chair can only be a thing of beauty to its owner and to his colonic cranks and anoerotic clientele. It therefore follows that a system of emptying the large intestine that is both reliably efficacious and capable of being carried out in the privacy of the patient's home deserves our close attention.

Overseas, diacetoxy-diphenyl-pyridyl-methane (dulcolax) has for some time been used for this purpose with considerable success. For sigmoidoscopy and barium enema all that is needed is for the patient to insert one dulcolax suppository into his rectum at 9 p.m. the night before the examination. He should lie down and be encouraged to hold it for at least a quarter of an hour; half an hour is better if he can. Then, at bedtime, he should swallow four dulcolax pills. Usually they will allow him to sleep until about 5 or 6 a.m. Finally, he should insert another suppository at about 7 a.m. By 9 a.m. he is safe to leave his house and he will have had from four to six good bowel openings and his colon will be cleaner than can be obtained by any other method. He can fortify himself with breakfast before he sets out.

For an IVP the patient does not need the full treatment. Four pills at bedtime are usually sufficient and they can be augmented by a matutinal suppository in the unlikely event of the dulcolax having failed to produce a satisfactory result. Twelve hours of no fluids—though a dry breakfast (eggs and toast) can be safely allowed—makes him ready for the radiologist. Both his costs and embarrassment have been kept to the minimum, his attendant specialists are contented and the possibility of water intoxication is wholly excluded.

KLINIESE ERVARING MET DIE KUNSNIER

L. F. MALHERBE, M.MED. (INT.), LEON JORDAAN, M.MED. (CHIR.), en G. E. J. SEYMORE, M.B., CH.B.

Departemente Interne Geneeskunde en Urologie, Algemene Hospitaal, Pretoria

DEEL I

Sedert Kolff sy proefskrif oor die kunsnier in 1946 gepubliseer het, het hierdie apparaat al hoe meer aandag in verskeie dele van die wêreld geniet. Tesame met die reeds welbeproefde konserwatiewe metodes van behandeling speel die kunsnier vandag 'n belangrike rol by die behandeling van gevalle met akute omkeerbare uremie. Die kunsnier is ook reeds van waarde gevind by die behandeling van sekere tipes vergiftiging, asook vir fisiopatologiese navorsing.

Die doel van hierdie artikel is om te dien as 'n weergawe van die kliniese ervaring wat ons opgedoen het met ekstrakorporeale hemodialise deur die gebruik van 4 verskillende soorte kunsniere. Nadat ons kunsmatige sowel as diereeksperimente uitgevoer het, het ons 41 hemodialises op 'n reeks van 26 pasiënte oor die afgelope 3 jaar gedoen.

Die grondbeginsels van die kunsnier is in die lewe geroep deur die eksperimente van Abel, Rowntree en Turner in1913.[1] Hulle het 'n selluloïde-buis as dialiseringsmembraan gebruik vir die verwydering van dialiseerbare stowwe uit die bloed van lewende diere. Tydens die eksperimente het hulle gebruik gemaak van hirudien om die bloed van hulle proefdiere vloeibaar te hou.

Sedert hierdie eksperimente het verskeie navorsers soos Necheles (1923), Haas (1935) en Thalheimer (1938) hierdie beginsel toegepas vir die verwydering van retensie-produkte uit die bloed van uremiese proefdiere sowel as pasiënte.[14,32]

Die eerste kunsnier wat klinies suksesvol gebruik is, is in 1943 deur Kolff en Berk in Nederland gebou.[13,11] Hulle het 'n sellofaanbuis as dialiseringsmembraan gebruik en het toe ook reeds oor heparien, wat 'n doeltreffende antistolmiddel is, beskik.

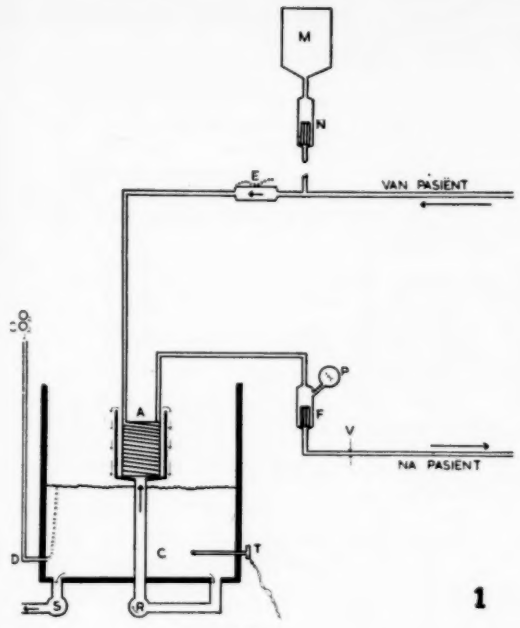
BEGINSELS VAN DIE KUNSNIER

Die beginsel waarop die werking van die kunsnier berus, is dialise, of differensiële diffusie van water en kristalloïdes, maar nie kolloïdes nie, deur 'n geskikte dialiseringsmembraan. Sellofaan word gewoonlik vir dié doel gebruik.

Afb. 1 is 'n diagrammatiese voorstelling van die konstruksie en metode van werking van 'n kunsnier.[16,17] Deur middel van 'n bloedpomp (E) word bloed vanaf 'n groot vena of arterie van die pasiënt, deur 'n geskikte dialiseringspoel (A) en 'n gekombineerde lugvanger en bloedfilter (F) gepomp en dan weer terug na die pasiënt. 'n Geskikte dialiseringspoel bestaan gewoonlik uit 'n lengte van sellofaanbuis wat in die vorm van 'n spoel gedraai is op so 'n manier dat bloed deur die lumen van die sellofaanbuis kan vloei.

Dialiseringsvloeistof word oor die buitekantste oppervlakte van die sellofaanbuis gesirkuleer. Verskuiwing van water en ander dialiseerbare stowwe kan dus plaasvind tussen die bloed in die lumen van die sellofaanbuis en die dialiseringsvloeistof aan die buitekant.

Die hidrostatiese druk van die bloed in die sellofaanspoel, kan beste bepaal word deur 'n manometer (P) te verbind met die lugruimte in die lugvanger aan die uitvloeikant van



Afb. 1.

die spoel.[16] Deur middel van 'n T-aansluiting aan die invloeikant van die spoel kan bloed of vog tot die sisteem toegevoeg word.

Om dialise meer doeltreffend te maak, is dit wenslik dat die totale dialiseringsoppervlakte groot genoeg is, terwyl die ingeslote bloedvolume so klein as moontlik gehou moet

TABEL I. FISIESE EIENSKAPPE VAN 'N KUNSNIER

	Mensnier	*Kunsnier (Sellofaan)*
Dialiseringsoppervlakte	7,600–1,500 vk. cm.	± 19,000 vk. cm.
Bloedvolume	10 ml.	± 1,000 ml.
Bloedvloei per minuut ..	1,250 ml.	± 250 ml.
Porië-deursnit	60–90 Å	60 Å
Porië-lengte	1 μ	80 μ
Ureumafsuiwering ..	75 ml./min.	± 180 ml./min.

word. Tabel I vergelyk die fisiese eienskappe van 'n kunsnier met dié van 'n mensnier.[24]

Die chemiese samestelling van die dialiseringsvloeistof kan verander word soos deur omstandighede vereis. Tabel II toon 'n formule aan wat bevredigend is en dikwels gebruik word. Die magnesiumgehalte is 1·5 m.Ekw./l. in plaas van 3 m.Ekw./l. soos oorspronklik deur Kolff *et al.* aanbeveel is.[16] Dit kan gesien word dat die elektrolietsamestelling by benadering soos dié van normale bloedplasma is. Deur glukose by te voeg word die osmolariteit van die dialiseringsvloeistof dan gebring tot dié van normale bloedplasma. Melksuur word by die dialiseringsvloeistof gevoeg totdat 'n pH van 7·4 bereik is en dan word 'n mengsel van 90% suurstof en 10% koolsuurgas deurgeborrel om die pH konstant by hierdie waarde te hou.

Deur middel van 'n elektriese verwarmer en termostat word die temperatuur van die dialiseringsvloeistof konstant gehou by 'n waarde net effens hoër as liggaamstemperatuur.

Die belangrikste faktore wat die verskuiwing van water, elektroliete en ander kristalloïdes deur die dialiseringsmembraan beïnvloed, sal later weer bespreek word, maar kan hier genoem word: (1) Die fisiese eienskappe van die dialiseringsmembraan (Tabel I), (2) die effektiewe hidrostatiese druk, (3) die effektiewe osmotiese krag, en (4) die ioon-konsentrasie gradiënt.

SOMMIGE KUNSNIERE REEDS GEBRUIK

Die oorspronklike Kolff-nier was die eerste kunsnier wat klinies suksesvol gebruik is. Die nier bestaan uit 'n horisontale drom waarom die sellofaanbuis in die vorm van 'n spoel gedraai is. Die drom roteer in 'n bad met dialiseringsvloeistof op so 'n manier dat net die onderste gedeelte daarvan deur die vloeistof beweeg. Enige bloed in die sellofaanbuis sak onder die invloed van swaartekrag na die onderste lusse van die sellofaanbuis en word voortbeweeg deur die rotasie van die drom. Die bloed word van en na die nier gepomp. Verskeie modifikasies van hierdie nier is reeds gebou.[14]

Alwall,[2] van Swede, het 'n apparaat ontwerp waar die sellofaanbuis om 'n vertikale, geperforeerde staalsilinder gedraai word. 'n Tweede silinder wat oor die eerste teleskoop, beperk die inwendige volume van die sellofaanbuis. Die dialiseringseenheid so verkry, word dan in 'n tenk met dialiseringsvloeistof geplaas. Deur die bloedtoevoer na die nier vanaf die pasiënt se arteria radialis te neem, het Alwall aanvanklik staat gemaak op die pasiënt se arteriële bloeddruk om die bloed deur die sellofaanspoel te dryf en het dus geen bloedpomp gebruik nie.

Uit Italië kom die nier van Battezzati en Taddei.[3] Die

TABEL II. DIALISERINGSVLOEISTOF

Sout	*G./l.*	*M.Ekw./l.*						*M.Osm./l.*
		Na^+	K^+	Ca^{++}	Mg^{++}	^-Cl	$^-HCO_3$	
NaCl	5·7	97·0				97·0		194·0
$NaHCO_3$	3·0	36·0					36·0	72·0
KCl	0·4		5·0			5·0		10·0
$CaCl_2$	0·28			5·0		5·0		7·5
$MgCl_2$	0·075				1·5	1·5		2·25
Glukose	4·0							22·2
		133	5·0	5·0	1·5	108	36·0	308
Normale Serum		142	4·5	5·0	1·5	103	28	311

1. Voeg glukose by tot gewensde osmolariteit.
2. Voeg melksuur by tot pH = 7·4.
3. Borrel 90% O_2 met 10% CO_2 deur vloeistof.
4. Verlaag KCl konsentrasie aan begin indien nodig.
5. Voeg $CaCl_2$ en $MgCl_2$ eers by na pH = 7·4.

dialiseringseenheid bestaan uit 3 vertikale sellofaanbuisspoele wat in 'n reeks verbind word. In hierdie nier word die sellofaanbuis tussen 'n binneste plastiese silinder en 'n buitenste plastiese omhulsel platgedruk om die volume van die sellofaanspoel te beperk. Die dele van die plastiese silinders en omhulsels wat in aanraking met die sellofaanbuis is, is gegroef op so 'n manier dat dialiseringsvloeistof aan die buitekant van die sellofaanbuis in 'n teenoorgestelde rigting as wat die bloed vloei, gepomp kan word. Die bloed word deur middel van 2 komplekse hidroliese rubberdiafragmapompe deur die 3 opeenvolgende spoele gepomp. Al 3 silinders staan regop in 'n plastiese tenk wat die dialiseringsvloeistof bevat.

Skeggs en Leonards[30] van Amerika het plat sellofaanvelle gebruik as dialiseringsmembrane. Die bloed word dan tussen die 2 sellofaanvelle deurgepomp in kanale wat ontstaan deurdat die sellofaanvelle tussen gegroefde rubber of plastiese plate vasgeklem word. Die dialiseringsvloeistof word dan oor die buitekant van hierdie kanale gesirkuleer.

Muirhead en Reid[26] het 'n kunsnier ontwerp wat met uitruilharse werk. Hierdie nier het nie algemene byval gevind nie.

Die nuutste ontwikkeling is 'n kunsnier wat in die Travenol Laboratoriums in Amerika ontwerp is na aanleiding van die werk wat gedoen is deur Inouye en Engelberg op 'n verbruikbare dialiseringseenheid.[16,17] Die dialiseringseenheid bestaan uit 2 sellofaanbuise wat tussen glasveselgaas in 'n spoel gedraai is. Die dialiseringseenheid word in 'n klein badjie oor 'n groter tenk aangebring en dialiseringsvloeistof word tussen die sellofaanlusse deurgepomp. Bloed word deur 'n sigmamotorpomp óf deur een óf deur albei sellofaanspoele gepomp. Die dialiseringseenheid, saam met die verbindingsbuise en pompbuise, word gesteriliseerd en gereed vir gebruik deur die vervaardigers verskaf.

In ons reeks gevalle het ons ondervinding opgedoen met 4 kunsniere, naamlik die apparaat van Battezzati-Taddei (2 modelle), die Alwall-apparaat, en die Travenol-nier.

UITWERKING VAN EKSTRAKORPOREALE HEMODIALISE OP DIE PASIËNT

Die wyse waarop ekstrakorporeale hemodialise 'n pasiënt beïnvloed, kan onder die volgende twee hoofde bespreek word: (A) Volumetriese veranderinge, en (B) chemiese veranderinge.

(A) Volumetriese Veranderinge

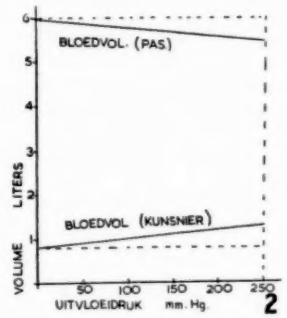
Gedurende ekstrakorporeale hemodialise vind sekere volumetriese veranderinge van die pasiënt se vog-kompartemente plaas. Volgens ons ondervinding is die belangrikste volumetriese veranderinge wat plaasvind, die volgende:

1. Onmiddellike bloedvolumeveranderinge te wyte aan die elastisiteit van die dialiseringsisteem.[15,18]
2. Filtrasie van water deur die dialiseringsmembraan met gevolglike hemokonsentrasie.[3,16]
3. Sedimentasie van bloedselle in die dialiseringsisteem met daling van die hematokritwaarde.
4. Meer permanente vogverskuiwings te wyte aan chemiese veranderinge wat volg op die diffusie van kristalloïdes.

1. *Die Elastisiteit van die Dialiseringsisteem*

Die uitvloeidruk en dus ook die gemene inwendige druk van die bloed in die dialiseringsisteem is afhanklik van etlike faktore. Die twee belangrikste faktore wat direk onder ons beheer staan tydens dialise is die *weerstand* aan die uitvloeikant van die sisteem en die *spoed* waarmee die bloed deurgepomp word.[16]

Aangesien die dialiseringsisteem nie absoluut onrekbaar is nie, is gevind dat die sisteem se inwendige volume op 'n nie-liniêre wyse toeneem met 'n vermeerdering van die inwendige druk. Aangesien die totale bloedvolume van die pasiënt plus die dialiseringsisteem as konstant beskou kan word oor kort tydperke, sal enige verandering van die inwendige volume van die dialiseringsisteem 'n resiprokale verandering in die pasiënt se bloedvolume veroorsaak.



Afb. 2.

Afb. 2 is 'n grafiese voorstelling van hierdie volumeveranderinge gekaart teenoor die uitvloeidruk, soos ons dit gevind het met die Travenol-kunsnier. Die praktiese implikasies van hierdie elastisiteit van volume is:

Eerstens is dit nodig om die dialiseringsisteem met bloed te laai onder 'n druk so na moontlik aan die druk wat gehandhaaf gaan word tydens dialise.[18] Nalating van hierdie voorsorgmaatreël het so 'n daling in bloeddruk by 2 van ons pasiënte veroorsaak, onmiddellik na die aanvang van dialise, dat ekstra bloed tot die sisteem toegevoeg moes word om die hipotensie te herstel.

Tweedens, nadat die dialise aan die gang is, moet skielike veranderinge van die snelheid van bloedvloei vermy word aangesien die inwendige druk, en dus ook die inwendige volume van die dialiseringsisteem, afhanklik is van die snelheid van bloedvloei. In ons reeks gevalle het ons tot dusver nog geen moeilikheid gehad as gevolg van hierdie feit nie, behalwe net verbygaande veranderinge in bloeddruk. Dit is egter moontlik dat skielike vermindering van bloedvloeisnelheid, veral by oorhidreerde anuriese pasiënte, longedeem mag veroorsaak.

2. *Filtrasie van Water deur die Dialiseringsmembraan*

Filtrasie van water deur die dialiseringsmembraan is afhanklik van: (*a*) Die fisiese eienskappe van die dialiseringsmembraan, (*b*) die effektiewe osmotiese druk, en (*c*) die effektiewe hidrostatiese druk.

(*a*) *Die fisiese eienskappe van die dialiseringsmembraan* is reeds bespreek. Soos in Tabel II aangetoon, is die elektrolietsamestelling van die dialiseringsvloeistof naastenby dieselfde as dié van normale bloedplasma. Om die osmolariteit van die dialiseringsvloeistof gelyk te maak aan bloedplasma, word glukose bygevoeg, gewoonlik tot 'n hoër konsentrasie as wat dit normaalweg in die plasma voorkom. Aangesien sellofaan deurlaatbaar is vir glukose, vind daar gedurende dialise 'n styging van die bloedglukosewaardes plaas.[15]

Die spoed van diffusie van glukose en die totale hoeveelheid glukose in die dialiseringsvloeistof is egter sodanig dat, tydens 'n gewone dialiseringstydperk, die glukose-konsentrasie in die dialiseringsvloeistof redelik konstant bly en hoër is as in die bloed. Om dieselfde redes bly die konsentrasie van ureum gewoonlik hoër in die bloed as in die badwater.

(*b*) *Die effektiewe osmotiese krag* wat die verskuiwing van water deur die sellofaanmembraan beïnvloed, word dus hoofsaaklik bepaal deur: Konsentrasie van plasmaproteïne, konsentrasie van bloedureum wanneer verhoog, en glukose-konsentrasie van die badwater.

(*c*) *Die effektiewe hidrostatiese druk* word ook deur etlike faktore bepaal waarvan die belangrikste soos volg is: Die metode en druk waarmee die bloed gepomp word, die snelheid van bloedvloei, die weerstand van die sellofaanspoel self, die weerstand van die uitvloeibaan, en die beginsel van negatiewe druk waar toegepas (*vide infra*).

Die effektiewe filtrasie-druk wat dus verantwoordelik is vir die filtrasie van water, kan soos volg gestel word:

Effektiewe filtrasie-druk = effektiewe hidrostatiese druk, *plus* effektiewe osmotiese krag van dialiseringsvloeistof, *minus* effektiewe osmotiese krag van bloed.

In die praktyk is hierdie faktore van variërende belang en afhanklik van die soort kunsnier wat gebruik word.

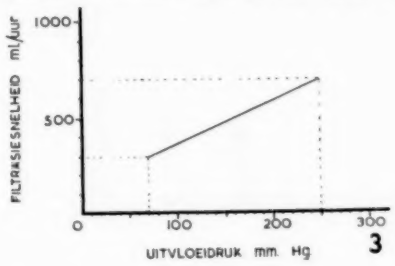
Met die roterende spoelnier (Kolff) is daar feitlik geen hidrostatiese druk nie en word die filtrasie van water hoofsaaklik deur osmotiese kragte bepaal.[15] In hierdie tipe nier kan die filtrasie van water in enige rigting deur die sellofaanmembraan plaasvind na gelang van die glukosekonsentrasie in die dialiseringsvog.

In die roterende spoelnier sowel as in sommige van die ander (bv. Alwall, Battezzati-Taddei) kan die filtrasiedruk vermeerder word deur die dialiseringseenheid in 'n lugdigte kompartement te plaas en negatiewe druk aan te wend.[3]

Met die gebruik van die Travenol-nier is die hidrostatiese druk soveel hoër as die effektiewe osmotiese druk dat dit die belangrikste enkelfaktor is wat die filtrasie van water bepaal.

Onder die invloed van bogenoemde faktore kan 'n pasiënt tot 6 liters of meer water verloor tydens 'n gewone dialiseringstydperk. Die neiging van hierdie vogverlies is om, onder andere, hemokonsentrasie met 'n styging van die hematokrit- en hemoglobienwaardes te veroorsaak. Verdere eksperimentele werk dui daarop dat vogverlies as gevolg van hidrostatiese filtrasie hoofsaaklik ekstrasellulêre dehidrasie veroorsaak, terwyl vogverlies te wyte aan osmotiese filtrasie hoofsaaklik deur sellulêre dehidrasie gevolg word.[28]

Om die pasiënt in behoorlike vogbalans te hou, moet die vogverlies tydens dialise vasgestel en vervang word nadat die toestand van hidrasie aan die begin van dialise in aanmerking geneem is. Hierdie vogverlies kan bepaal word deur gebruik te maak van: (i) die kliniese beeld tesame met 'n noukeurige rekord van die pasiënt se bloeddruk, polssnelheid, hematokrit- en hemoglobienwaardes; en (ii) die filtrasie-druk om die vogverlies te bereken.



Afb. 3.

Van die kunsniere wat ons tot dusver gebruik het, het ons die meeste ondervinding met die Travenol-nier. Soos reeds genoem, is die hidrostatiese druk by hierdie nier die belangrikste enkelfaktor wat die filtrasie van water bepaal. Met die gebruik van hierdie nier het ons die grafiek soos aangedui in Afb. 3 baie nuttig gevind. In hierdie grafiek is die filtrasiesnelheid van water teenoor die uitvloeidruk gekaart.[33] 'n Manometer, verbind met die lugruimte van die bloedfilter, gee die uitvloeidruk aan en die filtrasiesnelheid van water kan dan van die grafiek verkry word.

Tabel III (*a*) dui 'n geval aan waar die vogverlies gedurende dialise vervang is soos bereken vanaf bogenoemde grafiek.

TABEL III (*a*) EN (*b*). TOTALE VOG TOEGEDIEN TYDENS DIALISE

	Aan begin	*Na 2 uur*	*Na 4 uur*	*Na 6 uur*
(*a*)				
Bloeddruk	120/75	110/70	125/70	125/80
Polssnelheid per min.	128	130	132	128
Hematokrit	35%	36%	33%	—
Hemoglobien	74%	74%	73%	70%
Uitvloeidruk mm. Hg	150	190	190	190
Berekende vogverlies..	—	1,000 ml.	2,000 ml.	3,000 ml.
Totale vog toegedien..	—	900 ml.	1,700 ml.	2,900 ml.
(i) Bloed per 2 uur	—	400 ml.	500 ml.	1,000 ml.
(ii) 0.9% NaCl per 2 uur	—	500 ml.	300 ml.	200 ml.
(*b*)				
Bloeddruk	160/80	180/90	195/100	200/90
Polssnelheid per min.	86	96	96	104
Hematokrit	32%	35%	33%	34%
Hemoglobien	—	—	71%	—
Uitvloeidruk mm. Hg	90	100	110	100
Berekende vogverlies..	0	350	700	1,050
Totale vog toegedien..	0	0	500 ml.	500 ml.
(i) Bloed per 2 uur	0	0	500 ml.	0
(ii) 0·9% NaCl per 2 uur	0	0	0	0

Die bloeddruk en polssnelheid het feitlik konstant gebly gedurende die dialiseringstydperk.

Tabel III (*b*) dui 'n geval aan waar die vervanging van die berekende vogverlies onvoldoende was. By hierdie pasiënt, sowel as by ander soortgelyke gevalle, het ons gevind dat die bloeddruk en die polssnelheid gedurende dialise styg. Nog 'n groter agterstand in die vervanging van die vogverlies sal waarskynlik aanleiding gee tot 'n daling van die bloeddruk.

3. *Sedimentasie van Bloedselle*

Gedurende dialise vind sedimentasie van rooibloedselle in die dialiseringsisteem normaalweg plaas. Die gevolg hiervan is: (i) Daling van hematokrit- en hemoglobienwaardes, en (ii) vermindering van die pasiënt se bloedvolume.

Ons het die sedimentasie van rooiselle veral lastig gevind waar ons te doen gehad het met die laer bloedvloeisnelhede soos by die gebruik van die Alwall-nier sonder 'n bloedpomp.

As gevolg van die sedimentasie van rooiselle het ons dit sover nog altyd nodig gevind om 'n gedeelte van die vogverlies gedurende dialise met bloed aan te vul (Tabel III). Ons beskik nie oor 'n ander bevredigende metode om die rooiselverlies gedurende dialise vas te stel nie en gebruik dus herhaalde hematokrit- en hemoglobienwaardes as 'n leidraad by die bepaling van die hoeveelheid bloed wat toegedien moet word.

4. *Volumetriese Verandering as gevolg van Chemiese Manipulasie*

Hoewel die eerste effek van enige manipulasie van water, elektroliete of ander kristalloïdes in die vaskulêre kompartement gesien kan word, veroorsaak dit met tyd ook vogverskuiwings tussen die ander vogkompartemente van die liggaam.

'n Voorbeeld hiervan is die sellulêre dehidrasie wat volg op osmotiese filtrasie van water, terwyl hidrostatiese filtrasie ekstrasellulêre dehidrasie veroorsaak deur sowel ekstrasellulêre water as kristalloïdes te verwyder.[28] By pasiënte met edeem of oorhidreerde anuriese pasiënte kan sowel water as natrium dus effektief verwyder word deur dialise.

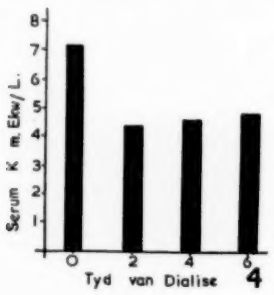
(B) Chemiese Veranderinge Tydens Dialise

Chemiese veranderinge wat plaasvind gedurende dialise kan bespreek word onder die volgende hoofde: (1) Manipulasie van elektroliete en ander kristalloïdes, (2) herstel van pH en alkalireserwe, (3) eliminasie van endogene en eksogene toksienes, en (4) verlies van aminosure, vitamines en ander stowwe.

1. *Manipulasie van Elektroliete*

Van Tabel II kan gesien word dat die elektrolietsamestelling van die dialiseringsvloeistof naasteby die patroon van normale bloedplasma volg. Die rigting waarin elektroliete en ander kristalloïdes deur die dialiseringsmembraan beweeg word deur hulle respektiewe konsentrasies in die bloedserum en dialiseringsvloeistof bepaal. Hierdie stowwe kan dus tot die bloed toegevoeg of daarvan verwyder word.

Daar bestaan veelvuldige kliniese sindrome van water en elektrolietwanbalans wat dikwels by uremiese pasiënte aangetref word. Die elektrolietsamestelling van die bloedserum, en dus ook die ekstrasellulêre vog, neig om die patroon van die dialiseringsvloeistof aan te neem en kan dus deur dialise na normale waardes teruggebring word. 'n Voorbeeld hiervan is die spoedige herstel van 'n normale serum kalium-



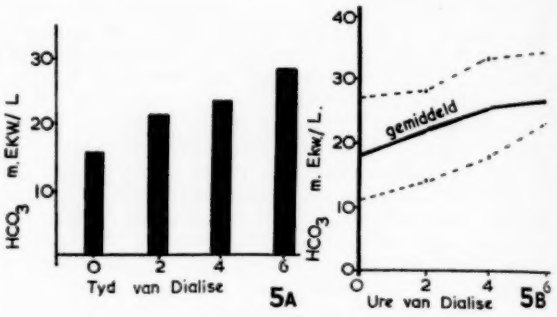
Afb. 4.

waarde tydens dialise (Afb. 4). By al ons gevalle met hiperkalemie het ons sover nog 'n bevredigende daling verkry met dialise.

Alhoewel hiperkalemie van uiters groot belang mag wees in die individuele geval, word dit by die minderheid van oliguriese of anuriese pasiënte waargeneem.[9,11,29] In teenstelling hiermee is 'n styging in die bloedhoogte van magnesium en sulfate 'n konstante waarneming by alle anurie-pasiënte. Die magnesium-, sulfaat-, en fosfaatwaardes word vinnig na normaal gebring deur hemodialise terwyl peritoneale dialise nie so 'n gunstige uitwerking het nie.[9-11,29]

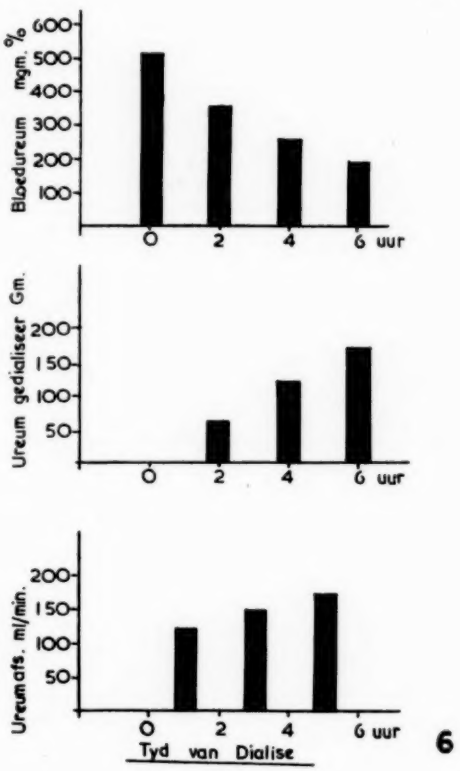
2. *Herstel van Alkalireserwe en pH*

Deur verwydering van oormatige suurione en herstel van die serum elektrolietpatroon, veroorsaak hemodialise 'n spoedige verbetering van die alkalireserwe by pasiënte met uremie en metaboliese asidose. Afb. 5 (*a*) toon 'n mooi



Afbs. 5A en B.

voorbeeld hiervan soos ons dit by 'n pasiënt in ons reeks gevind het. Afb. 5(*b*) toon die gemiddelde waardes van HCO_3 by 23 dialises met die Travenol-nier aan. Tot dusver het ons nog nie serum pH-bepalings gedoen nie, maar het gevind dat die kliniese beeld van asidose goed verbeter gedurende dialise.



Afb. 6.

3. *Verwydering van Toksiese Stowwe*

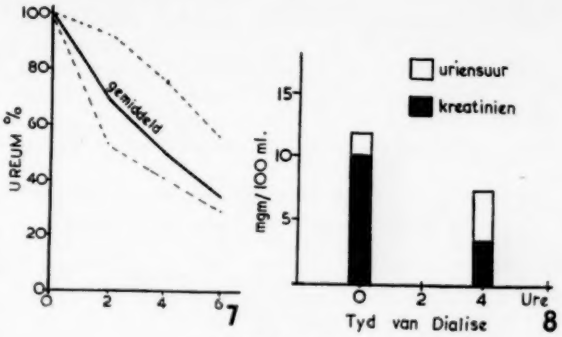
Behalwe vir die dialiseerbare metaboliese sure kan verskeie toksiese stowwe van eksogene sowel as endogene oorsprong deur dialise van die liggaam verwyder word.

Wat die eksogene toksienes betref, is reeds in 1914 deur Abel, Rowntree en Turner aangetoon dat salisilate dialiseerbaar is. Dit is nou bevestig dat pasiënte wat vergiftig is met salisilate,[6,17] bromiedes,[21] fenobarbitoon, en tot 'n mindere mate pentobarbitoon, goed reageer op hemodialise. Secobarbitoon en amobarbitoon verbind met die bloedproteïne en is feitlik nie dialiseerbaar nie.[15,17,19] Streptomisien-[7] en ammoniak-[12]vergiftiging kan ook deur dialise verbeter word. Ons het nog nie sulke gevalle gedialiseer nie.

Sommige toksienes van endogene oorsprong kan ook deur dialise beïnvloed word. Ureum, hoewel nie as toksies beskou nie, word verwyder gedurende dialise en kan dien as 'n maatstaf om die vordering van die dialise aan te toon. Onder sekere omstandighede word ureum by die dialiseringsvloeistof gevoeg en kan die verwydering van ander res-stikstofprodukte soos uriensuur of kreatinien vir hierdie doel gebruik word.

Afb. 6 stel die verloop van 'n dialise met die Travenol-nier in ons reeks grafies voor soos aangetoon deur die bloedureumgehalte, totale ureum van die pasiënt verwyder, en berekening van die ureumafsuiweringswaarde van die kunsnier. Afb. 7 toon die gemiddelde bloedureumwaardes tydens 23 dialises met die Travenol-nier aan. Om vergelykbare waardes te kry, is die bloedureumgehalte uitgedruk as 'n persentasie van die aanvangswaarde by elke pasiënt. Afb. 8 toon hoe die bloeduriensuur en kreatinienwaardes verander het tydens 'n dialise in ons reeks.

Daar bestaan nog die moontlikheid van onbekende toksiese stowwe wat deels verantwoordelik is vir die kliniese sindroom van uremie. Indien daar wel sulke toksiese stowwe



Afbs. 7 en 8.

bestaan, moet ons aanneem dat hierdie stowwe ook dialiseerbaar is aangesien die beeld van akute uremie so dramaties verbeter tydens dialise.

4. *Verlies van Aminosure en Vitamiene*

Dat aminosure dialiseerbaar is, is reeds in 1913 deur Abel, Rowntree en Turner aangetoon. Ons het gevind dat die konsentrasie van aminosure in die dialiseringsvloeistof na 'n gewone dialiseringstydperk nog te laag is om direk met papierkromatografie te bepaal. Geringe hoeveelhede proteïen is soms aantoonbaar in die dialiseringsvloeistof, maar ons het nog nie kwantitatiewe bepalings hierop uitgevoer nie.

Ons dien gewoonlik vitamiene van die B-kompleksgroep, asook vitamien C, aan ons anurie-pasiënte toe weens die moontlikheid dat 'n tekort aan hierdie stowwe deur dialise, asook gebrekkige inname, by hierdie pasiënte mag ontstaan.

(Word vervolg.)

SKIN DISEASE IN THE WHITE SOUTH AFRICAN

A SURVEY OF THE INCIDENCE OF SKIN DISORDERS IN 13,500 DERMATOLOGICAL PATIENTS FROM THE TRANSVAAL AND ORANGE FREE STATE

G. H. FINDLAY, M.D., *Section of Dermatology, University of Pretoria*, and F. P. SCOTT, M.MED., *Dermato ogist, National Hospital, Bloemfontein*

No publication exists on the frequencies of skin disorders in the White South African, and we feel that the present survey needs no apology. The material comprises 3 groups of consecutive patients, one from the Pretoria Hospital skin outpatient service (4,500) and the other two from the private consultant practices of each of us (5,000—G.H.F., 4,000—F.P.S.). All the diagnoses in the Pretoria Hospital group were made by one or other of the writers, except for some 1,500 seen by our assistants, Drs. I. J. Venter and E. J. Schulz.

Most of the patients in all 3 groups were Afrikaans-speaking. The hospital group was gathered from poorer people, and minor skin ailments were more readily referred to us by other sections of the hospital than they would be in private practice. At hospital there were also more patients who would in private practice sooner find themselves in the hands of a surgeon or radiotherapist.

The results in the 3 groups of our series are set out in the accompanying table together with those in the 4 other groups referred to below, which are included for the purpose of comparison.

With a few exceptions, no condition with a frequency below 0·1 % is noted in the table. The sequence of the diseases listed is intended to follow roughly the frequencies met with in South Africa, and thus to reflect their relative importance to the practitioner. It should be noted, however, that some important disorders (e.g. malignant melanoma) are too rare to find a place in the list.

Comparative figures are not easily accessible, and for this reason we have quoted the findings from three other sources, re-calculated to permit direct comparison. *The Australian series* presented here, incomplete as it is, provides interesting information from the Southern Hemisphere where White people also live a sunny outdoor life. It is adapted from a paper by Summons.[1] *The London figures* of Calnan and Meara[2] from St. John's Hospital are doubtless the best that have appeared from Britain, and are an invaluable source for

comparative studies. *The third and fourth series* included here for comparison are compiled from the Appendix in Radcliffe-Crocker's classic text-book.[3] They give one an idea of conditions and practice in London at the turn of the century. One gathers that the surgeons claimed a greater share of skin disorders at that time than now. With the growth in the operative aspects of surgery, the dermatologist appears to have taken over some of these disorders.

REFERENCES

1. Summons, J. (1955): Austral. J. Derm., 3, 15.
2. Calnan, C. D. and Meara, R. H. (1957): Trans. St. John's Hosp. Derm. Soc. (Lond.), 39, 56.
3. Radcliffe-Crocker, H. (1905): *Diseases of the Skin*, 3rd ed., vol. 2. London: H. K. Lewis.

PERCENTAGE ENUMERATION OF CASES IN DERMATOLOGICAL PRACTICE

I Pretoria General Hospital, skin out-patients, Transvaal, South Africa, 1956–59. 4,500 cases seen by 4 dermatologists (see text).
II Private dermatological practice (G.H.F.), Pretoria, Transvaal, South Africa, 1955–59. 5,000 cases.
III Private dermatological practice (F.P.S.), Bloemfontein, Orange Free State, South Africa, 1956–59. 4,000 cases.
IV Private practice of 4 dermatologists, in Melbourne, Victoria, Australia, 1938–52. 25,296 cases.
V St. John's Hospital for Diseases of the Skin, London, England, 1952–56. 90,000 cases seen by 20 consultants.
VI University College Hospital (skin out-patients), London, England, *c.* 1900. 10,000 cases. Radcliffe-Crocker.
VII Private dermatological practice (Radcliffe-Crocker), London, England, *c.* 1900. 5,000 cases.

		I	*II*	*III*	*IV*	*V*	*VI*	*VII*
1	Eczemas(*a*)	28%	35%	29·4%	27·3% + 5·66%(*v*)	±35%	27·2%	26·1%
2	Acne vulgaris(*b*)	8%	10%	11·3%	4·85%	5·7%	2·5%	6•2%
3	Impetigo	7·8%	2·2%	3%	4·78% (*incl.* 12 *and* 43)	1·9%	9·6%	1%
4	Fungous infections(*c*)	6·9%	3·3%	5·3%	2·59%	2·4%	13·1%	3·1%
5	Warts(*d*)	5·9%	4%	4·2%	11·78%	7%	? nil or 0·1%	0·9%
6	Rodent ulcer	4·5%	2·2%	2·5%	7·88% (*incl.* 24)	0·87%	0·1%	0·7%
7	Psoriasis	3·2%	4·6%	4·8%	2·57%	5·6%	7·2%	6%
8	(*a*) Senile and solar keratoses	2·1%		4%	4·95%	0·1%	? nil	nil
	(*b*) Seborrhoeic warts	1%	3·6%	1%	—	0·5%	or 0·1%	or ? 3 cases
9	Urticaria(*e*)	2·3%	2·7%	1·2%	1·08%	1·7%	4·4%	3·8%
10	Scabies	2·5%	1·1%	1·6%	0·08%	0·8%	8%	1·2%
11	Sandworm(*f*)	2·4%	1·2%	0·4%	—	nil	nil	nil
12	Pyodermas(*g*)	1·6%	0·5%	0·45%	*see* 3	0·8%	?	?
13	Pityriasis rosea	1%	2·3%	1·3%	0·8%	1·5%	0·4%	1·2%
14	Light eruptions(*h*)	1%	1·5%	1·6%	12·8%(*w*)	0·4%	?	0·4%
15	Varicose ulcer	1·2%	0·3%	2%	?	0·41%	nil	nil
16	Lichen urticatus	1·2%	0·7%	1·2%	?	0·5%	?	?
17	Furuncle, carbuncle	1·1%	0·4%	0·9%		0·5%	0·35%	0·6%
18	Moles (cellular naevi)	1·1%	1·1%	1·3%		1·2%	nil	0·4%
19	Rosacea	0·2%	1%	0·4%	1·73%	1·4%	2%	6%
20	Puritus ani and vulvae	0·4%	1·1%	0·8%	1·8% *incl.* 37	1·1%	? *incl. in* 37	1·6%(*bb*)
21	Ichthyosis(*i*)	0·3%	1·1%	0·85%		0·3%	0·54%	0·7%
22	Lupus erythematosus(*d*)	0·9%	1%	0·8%		0·4%	0·6%	1·8%
23	Lichen planus	0·5%	0·9%	0·4%	0·3%	1·3%	1%	2·3%
24	Squamous carcinoma	0·7%	0·4%	0·8%	*see* 6	0·08%	nil	0·16%
25	Vitiligo	0·4%	0·7%	0·9%		0·4%	0·2%	0·4%
26	Erythema multiforme	0·6%	0·7%	0·4%		0·46%	1·1%	1·3%
27	Alopecia areata	0·6%	1·2%	1%		1·9%	2·5%	4·8%
28	Corns and calluses	0·6%	0·36%	0·2%		0·2%	nil	0·2%
29	Excoriations and artifacts	0·3%	0·5%	0·1%		0·2%	4 cases	nil
30	Haemangioma(*d*)	0·5%	0·5%	0·4%	? 2·7%	0·9%	3 cases	0·24%
31	Leukoplakia	0·5%	0·4%	0·8%		0·05%	nil	ni
32	Toxicodermas(*j*)	0·2%	0·6%	1%		0·6%	0·1%	0·3%
33	Herpes zoster	0·6%	0·3%	0·4%		0·2%	0·6%	0·4%
34	Erythema nodosum	0·5%	0·4%	0·1%		0·02%	nil	nil
35	Pityriasis versicolor	0·4%	0·7%	0·45%		0·2%	0·3%	0·3%
36	Prurigo group(*k*)	0·2%	0·3%	*incl.* in 16		0·1%	0·2%	nil
37	Prutitus (various)	0·6%	0·3%	0·6%	*see* 20	0·9%	0·9%	0·8%
38	Granuloma annulare	0·2%	0·4%	0·4%		0·14%	nil	2 cases
39	Herpes simplex	0·3%	0·3%	0·2%		0·4%	0·5%	0·4%
40	Pernio, cold injury	0·3%	0·1%	0·15%		0·2%	? nil	? nil
41	Hyperidrosis	0·2%	0·3%	0·35%		0·2%	0·1%	0·3%
42	Exfoliative dermatitis	0·1%	0·3%	0·1%		0·03%	0·14%(*z*)	0·3%
43	Folliculitis and sycosis barbae	0·6%	0·5%	0·3%	*see* 3	2·12%	0·8%	1·1%
44	Chloasma(*l*)	0·1%	0·4%	0·5%		0·3%	2 cases	0·2%
45	Hairfall(*m*)	0·1%	0·4%	0·25%		1·3% (?)	5 cases	9·8%
46	Molluscum contagiosum	0·2%	0·2%	0·1%		0·2%	0·2%	0·2%
47	Porphyria	0·2%	0·2%	0·2%	?	2 cases	?	?
48	Milia	0·1%	0·2%	0·1%		0·1%	1 case	2 cases
49	Cysts (mainly sebaceous)	0·2%	0·1%	0·8%		0·2%	—	2 cases
50	Cornu cutaneum	0·2%	0·2%	0·35%		0·07%	? nil	? nil
51	Kerato-acanthoma(*n*)	0·1%	0·1%	0·25%	?	0·2%	?	?
52	Keloid	0·1%	0·1%	0·05%		0·1%	2 cases	0·2%

		I	*II*	*III*	*IV*	*V*	*VI*	*VII*
53	Intertrigo (uncomplicated)(*o*) ..	0·1%	0·1%	0·25%	?	0·54%	?	?
54	Chondrodermatitis helicis ..	0·1%	0·1%	1 case	?	0·03%	?	?
55	Colloid milium	0·1%	0·1%	0·1%	?	nil	?	?
56	Radiodermatitis	0·1%	0·1%	0·25%		0·06%	nil	1 case
57	Telangiectases	0·1%	0·1%	0·1%		0·1%	3 cases	0·9%
58	Fevers(*p*)	0·1%	0·1%	0·05%		0·04%	0·4%	nil
59	Parasitoses(*q*)	0·2%	0·1%	1 case	± nil(*x*)	0·1%	3·9%	2·3%
60	Fibroma	0·2%	0·2%	0·2%		0·02%	1 case	3 cases
61	Cheilitis(*d*)	0·2%	0·3%	0·3%		0·1%	?	?
62	Keratosis pilaris	0·2%	0·2%	0·1%		0·06%	nil	nil
63	Allergide, etc.(*r*)	0·3%	0·3%	0·3%		? 0·2%	0·1%(*aa*)	3 cases
64	Granuloma pyogenicum	0·1%	0·2%	0·4%		0·2%	nil	nil
65	Keratolysis exfoliativa	0·1%	0·5%	—		?	?	?
66	Erysipelas and erysipeloid(*s*) ..	0·1%	0·2%	3 cases		0·02%	nil	nil
67	Nail dystrophies	0·2%	0·3%	0·2%		0·3%	0·2%	0·7%
68	Cellulitis	0·2%	—	—		0·05%	nil	nil
69	Lichen sclerosus et atrophicus ..	0·1%	0·1%	0·1%		0·05%	—	—
70	Dermatitis herpetiformis	0·1%	0·1%	0·15%	0·2%	0·1%	0·1%	0·6%
71	Scleroderma	0·1%	0·1%	3 cases		0·1%	8 cases	0·2%
72	Lipid proteinosis	13 cases(*t*) (0·1%)				nil	—	—
73	Pellagra	7 cases(*t*)				nil	—	—
74	Tuberculides	5 cases(*t*)				0·06%	—	—
75	Leprosy	2 cases(*t*)				0·06%(*y*)	6 cases	0·3%
76	Pemphigus	4 cases(*t*)			0·06%	0·03%	0·3%	0·3%
77	Pseudoxanthoma elasticum ..	8 cases(*t*)				4 cases	—	—
78	Sporotrichosis	4 cases(*t*)(*u*)			—	nil	—	—
79	Chromoblastomycosis	2 cases(*t*)(*u*)			?	nil	—	—
80	Sarcoid Boeck	1 case(*t*)			?	11 cases	?	?
81	Syphilis	2 cases(*t*)			16 cases	29 cases	5·4%	2·6%
82	Lupus vulgaris	1 case(*t*)			?	53 cases	1·3%	1%

(*a*) Including infantile, atopic, constitutional, seborrhoeic, pompholyx, contact, eczematide, varicose, chronic, infective, pityriasis alba, traumatic, seasonal, neurodermatitis, lichen simplex chronicus, and unclassified; excluding solar eczema.
(*b*) And related types.
(*c*) Including tineas and moniliasis; excluding pityriasis versicolor.
(*d*) All types.
(*e*) Excluding lichen urticatus—see 16.
(*f*) Larva migrans.
(*g*) Including ecthyma, abscess, paronychia, etc.; excluding impetigo, boils, carbuncles.
(*h*) Solar eczema, etc.
(*i*) Severe xerosis.
(*j*) Including drug eruptions.
(*k*) Excluding Besnier type.
(*l*) And allied disorders.
(*m*) Excluding alopecia areata.
(*n*) Molluscum sebaceum.
(*o*) Uncomplicated.
(*p*) All types with exanthem.
(*q*) Including lice, myiasis, ticks, cercaria, and other insect bites.
(*r*) Including capillaritis, itching purpura, and Schamberg's disease.
(*s*) Frequency about equal.
(*t*) In 13,500 total.
(*u*) All from Transvaal.
(*v*) Seborrhoeic eruptions.
(*w*) Chronic solar dermatitis.
(*x*) Pediculosis.
(*y*) ? Whites.
(*z*) 'Pityriasis rubra'.
(*aa*) 'Purpura'.
(*bb*) 'Local pruritus'.

GASTRIC MUCORMYCOSIS : REPORT OF CASE IN A SWAZI

JOHN C. SUTHERLAND, M.D. (MARQUETTE), and T. HAROLD JONES, M.B., B.CH. (RAND)

The Ethel Lucas Memorial Hospital, Acornhoek, Eastern Transvaal

Fungi such as the aspergilli, penicillia and mucors that were once familiar chiefly as laboratory contaminants are sometimes the cause of severe and often fatal disease. This is perhaps part of a changing pattern of disease effected by antibiotic-, steroid- and chemotherapy. Cases of mucormycosis are being reported in increasing numbers from the Americas and Europe. Usually they occur secondarily to some other disease, especially uncontrolled diabetes and leukaemia. Cortisone, broad-spectrum antibiotic and antileukaemic therapy may be predisposing factors.[1] The following case is the second to be published from South Africa although several others have been seen at the South African Institute for Medical Research in Johannesburg,[2] at the University of Natal,[3] and possibly elsewhere.

CASE REPORT

J.D., a Swazi man of about 50 years, came to the hospital on 31 May 1958 because of cough, blood-stained sputum, and sharp pain in the chest which began 3 weeks previously. Occasionally there was postprandial abdominal pain. The patient was weak and emaciated. Moist rales could be heard in both lungs and there was a palpable epigastric tumour. The temperature was subnormal. The urine was not tested. 600,000 units of procaine penicillin were given after admission and again the following morning. The patient died early in the morning of the third day in hospital. The clinical diagnosis was pulmonary tuberculosis and carcinoma of the stomach.

An autopsy was performed 48 hours *post mortem*. The principal findings were in the lungs and stomach. The left lung was dark, heavy and firm and from the cut surface a frothy haemorrhagic fluid could be expressed. No cavities were noted. The lower lobe of the right lung was similar to the left but in a lesser degree. Within the stomach there was a greyish-white tumour, about 3 × 2 inches, along the greater curvature near the cardia. The surface of this tumour was elevated about 3/8th inch above the surrounding mucosa and was covered with a gelatinous exudate. The remainder of the gastro-intestinal tract was normal by palpation, but was not opened. Sections of the stomach, lungs, liver, one kidney and aorta were put in 10% formalin and sent to the Armed Forces Institute of Pathology in Washington, D.C., whose report, by Elson B. Helwig, M.D., was as follows:

'The lesion of the stomach is composed of necrotic and haemorrhagic material which is diffusely infiltrated by numerous and non-septate hyphae typical of mucormycosis (Fig. 1). Vascular invasion, a characteristic feature of mucormycosis is also prominent in this case (Fig. 2). Since this fungus is not liable to penetrate intact mucosa it is likely that the infection followed some mucosal defect, possibly a gastric ulcer. Diligent search of sections of other organs did not yield any evidence of systemic spread by the fungus. Only one other case of gastric mucormycosis, very similar to the present one and contributed from Korea, is on file at the A.F.I.P. In addition, sections of the liver and lung show evidence of schistosomiasis; several ova with minimal tissue reaction are present, but are not sufficiently characteristic

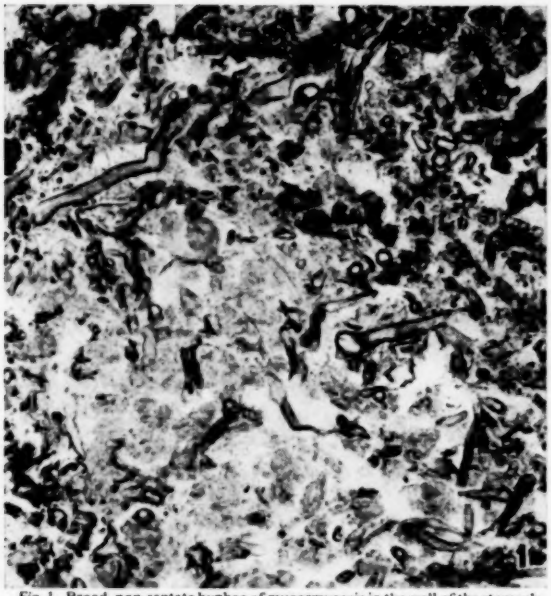


Fig. 1. Broad, non-septate hyphae of mucormycosis in the wall of the stomach, H & E, × 380. (*Armed Forces Institute of Pathology, Washington.*)

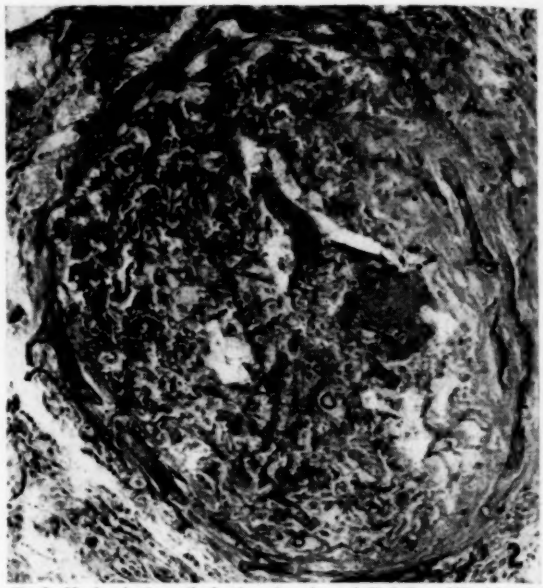


Fig. 2. Hyphae in the wall of a thrombosed vessel, H & E, × 330. (*Armed Forces Institute of Pathology, Washington.*)

to allow any further classification. Both lungs exhibit a severe and necrotizing pneumonitis. The aorta shows mild arteriosclerotic changes with thickening of the subintimal tissue, but does not manifest any evidence of syphilis.'

A nephew, contacted after the patient's death, said that the patient had been ill with a chronic cough for 3 years. He was fond of Bantu beer, especially *ngodwana*, or strong beer, and his family attributed his illness to this habit. During the last 3 years he worked as a policeman on various farms in the Lowveld. He never worked in the mines. His nephew last saw him 1 year before he died.

DISCUSSION

Mucormycosis is infection by fungi of the family Mucoraceae, which includes 3 genera, viz. Mucor, Absidia and Rhizopus.[4] They are ubiquitous, occurring as saprophytes on dead or decaying plants and animals and they often contaminate laboratory cultures.[5] In mucormycosis the characteristic histological finding is the presence of large, non-septate hyphae in arterial walls, associated with thrombosis and infarction.[1] It is necessary to culture the organism to decide in which genus it belongs and in the present case the identification cannot be refined beyond that of one of the Mucoraceae.

The most frequent sites for mucormycosis are the lungs and brain.[1] In the lungs there may be bronchopneumonia[6] or a lung abscess.[7] Both air-borne and haematogenous infections may occur.[1] In the brain the lesion is that of meningo-encephalitis, often with ophthalmoplegia, and usually found in the presence of uncontrolled diabetes.[8,9] Thrombosis of the lateral sinus has occurred.[2] The route of infection is probably from the nose *via* the paranasal sinuses or through the cribriform plate or from the eyes.[1,9]

Reports of gastro-intestinal mucormycosis in the literature are scanty so that as late as 1957 Baker could review only 5 cases, including 2 of his own.[10] Usually there is haemorrhagic necrosis and ulceration of the intestinal wall, sometimes with perforation.[5,10,11] Tumour-like lesions similar to the present case have been reported.[12] Mesenteric thrombosis may occur. All portions of the gastro-intestinal tract, including the oesophagus, may be involved. The clinical features are those of an ulcerative colitis, peritonitis and intestinal obstruction. Embolic abscesses may occur in the liver.[10,12] In none of the cases reviewed by Baker *et al.*[10] was the fungus noted clinically in the stool. The previous South African case is one of gastric mucormycosis in a 26-month-old Bantu child suffering from advanced kwashiorkor.[13] Malnutrition was also a prominent feature in another South African case, a Bantu infant,[2] and in 2 Brazilian cases, aged 10 months and 1 year.[11]

Gastro-intestinal mucormycosis may occur in the absence of an antecedent disease and is usually considered to be caused by ingested spores.[10] It is interesting to speculate on the role of *ngodwana* in this case. This is the Shangaan name for strong beer which is made as follows. Corn meal is mixed with water and allowed to stand for a week or 10 days. Then it is cooked in the manner of porridge, cooled and thinned with water. Kaffir corn (*Sorghum vulgare*) which has previously been allowed to sprout and then dried and partially ground is added with sugar to the corn-meal mixture. This final mixture is allowed to ferment 2 or 3 days, when drinking usually begins. With such favourable conditions for the growth of a fungus, perhaps many spores are ingested in drinking *ngodwana*. Fermented foods might have been a factor in other South African cases. The soft porridge given to Bantu children is usually cooked after fermenting. Sometimes, however, it may ferment after cooking (*amahewu*). The 2 Brazilian cases were also associated with malnutrition. The staple diet of the lower classes in Brazil is low in protein and rich in carbohydrates. Fermentation, however, is not a part of its preparation.[14]

Ingested spores are thought incapable of penetrating

intact mucosa and the possibility of a gastric ulcer in this case is suggested. In the absence of an adequate history we can only speculate. Recent autopsy studies in Uganda suggest a higher incidence of both gastric and duodenal ulcers amongst the Bantu than was previously suspected.[15]

Death in this case was probably caused by pneumonia. We are unable to say whether or not this preceded the gastric infection. Clark suggests that the presence of a tumour-like mass in gastro-intestinal mucormycosis may signify an infection of longer duration than usually occurs.[12]

An additional feature was the bilharzial ova in the liver and lungs. Bilharziasis is common here in the Lowveld. Dissemination of the ova probably decreased the patient's resistance and may have been a contributory factor in the fungus infection.

The clinical diagnosis of mucormycosis is not easy. In most of the reported cases an antemortem diagnosis was not made, perhaps because it was not suspected. At least 6 cases reported since 1955, however, have recovered.[9] Of these, 5 were cranial cases and the diagnosis was confirmed either by a culture or by a biopsy in 4 of them. The 6th was a pulmonary case, in which a lobectomy was done for lung abscess.[7] All 6 cases were in uncontrolled diabetics.

The possibility of mucormycosis should be kept in mind in any severe disease that is being intensively treated with antibiotics, steroids or antileukaemic agents. Also, in such a disease, worsening of the patient's condition should arouse suspicion that a fungus infection has possibly occurred. To confirm the diagnosis, cultures on Sabouroud's medium can be made from sputum, bronchial washings, nasal smears, gastric contents, stool, cerebrospinal fluid and the exudate from chronic ulcers. Biopsies may be taken from suspicious lesions. Laboratory contamination of cultures is likely and the spores of these fungi may be found in the nose, sputum and stomach of healthy persons. Thus close co-operation is necessary between clinician and mycologist to assess the significance of positive cultures.[5]

The treatment of mucormycosis is difficult. If diabetes is present, the hyperglycaemia should be carefully controlled and all steroid and antibiotic therapy stopped or at least re-evaluated.[1] Diabetic control was the chief feature in the 6 recovered cases. The following have also been used in one or more of these cases: potassium iodide, desensitization to the fungus, parenzyme, antibiotics and mycostatin.[9] The focus of infection was excised in 2 cases. The use of antibiotics in the treatment of 1 case, and also mycostatin is interesting, although of doubtful value. In another report the use of amphotericin B is suggested.[11]

SUMMARY

A case of gastric mucormycosis in a Swazi man, associated with a necrotizing pneumonitis, is presented. Mucormycosis, especially of the lungs and brain, usually occurs secondarily to some other disease such as uncontrolled diabetes and leukaemia. Gastro-intestinal mucormycosis is less often seen and may occur primarily. The fermented foods and beer of the Bantu may possibly be factors in gastro-intestinal fungus infections by the introduction of excessive numbers of spores.

We wish to thank Dr. H. I. Lurie for his helpful suggestions and the Armed Forces Institute of Pathology, Washington, for the photomicrographs.

REFERENCES

1. Baker, R. D. (1957): J. Amer. Med. Assoc., **163**, 805.
2. Lurie, H. I.: Personal communication.
3. Watson, K. D.: Personal communication.
4. Smith, D. T. (1957): J. Chron. Dis., **5**, 528.
5. Moore, M., Anderson, W. A. D. and Everett, H. H. (1949): Amer. J. Path., **25**, 559.
6. Stefanini, M. and Salvatore, A. (1957): New Engl. J. Med., **256**, 1026.
7. Dillon, M. L., Sealy, W. C. and Fetter, B. F. (1958): J. Thorac. Surg., **35**, 464.
8. Bauer, H., Ajello, L., Adams, E. and Hernandez, D. U. (1955): Amer. J. Med., **18**, 822.
9. Long, E. L. and Weiss, D. L. (1959): *Ibid.*, **26**, 625.
10. Baker, R. D., Bassert, D. E. and Ferrington, E. (1957): A.M.A. Arch. Path., **63**, 176.
11. Montenegro, M. R., Brito, T. de, Lombardi, J. and Lacaz, C. da S. (1959): Rev. Hosp. Clin. Univ. S. Paulo, **14**, 59.
12. Clark, R. M. (1957): Gastroenterology, **33**, 985.
13. Watson, K. C. (1957): S. Afr. Med. J., **31**, 99.
14. Lacaz, C. da S.: Personal communication.
15. Raper, A. B. (1958): Trans. Roy. Soc. Trop. Med. Hyg., **52**, 535. Quoted in Editorial (1959): Brit. Med. J. (1959): **1**, 1462.

DUPLICATION OF THE ALIMENTARY TRACT : REPORT OF A CASE

J. E. ROOS-SCHOLTZ, M.B., CH.B., F.R.C.S.E., *Pretoria*

A 4-month-old White male child was admitted to the Pretoria General Hospital with a history, given by the mother, of diarrhoea and vomiting for a week before admission. The vomiting occurred after each feed, it was not projectile in nature, and the vomited material consisted only of ingested material. The stool was green in colour, copious and watery, but was never blood-stained. The child had been feverish and irritable but never definitely suffering from cramp-like pains or abdominal distension.

The child was found to be well fed though slightly anaemic but with no jaundice or dehydration. The pulse rate was rapid and the temperature 100·8 F on admission. The abdomen was not distended but a mass was palpable in the right iliac fossa which was thought to be an ileocaecal intussusception, being sausage-shaped and lying longitudinally in the lower abdomen. On rectal examination the mass could be palpated on the right side and it was found to be mobile on bimanual manipulation.

X-ray examination with a barium enema was carried out and, though the terminal part of the ileum was not filled, the rest of the colon filled easily and quickly and was normal in outline. The caecum was seen to be displaced medially. Excretion and retrograde pyelography showed the kidneys to be normally situated, thus excluding a tumour or anomaly of renal origin. The intestinal gas shadows were seen to be displaced medially in the right lower abdomen. The diagnosis of mesenteric cyst was made, and the attacks of diarrhoea and vomiting were thought to have been caused by gastro-enteritis.

The gastro-enteritis cleared up with regulation of the diet and oral antibiotics, and the anaemia was corrected by blood transfusion.

At operation, under general anaesthetic through a right lower abdominal paramedian incision, a cystic tumour measuring about $2\frac{3}{4} \times 1\frac{3}{4} \times 1\frac{1}{2}$ inches, and dumb-bell shaped, was seen lying longitudinally alongside and lateral to the caecum. The appendix was directed laterally and its tip was firmly adherent to the anterior aspect of the lower pole of the cyst. A tail of omentum was adherent to the waist, and from both the omentum and the appendix blood vessels radiated over the cyst wall. A branch of the right colic artery passed behind the ascending colon and spread over the upper pole of the cyst (Fig. 1). Removal of the cyst was accomplished by detaching the appendix from the caecum and ligating and invaginating the stump; dividing the omentum about $1\frac{1}{2}$ inches above its extremity, and ligating the branch of the right colic artery to the cyst. The cyst was easily separated from the caecum. The abdomen was closed in layers without drainage of the peritoneal cavity. Convalescence was uneventful and the child was allowed home on the 7th post-operative day.

Anatomy and histology. The dumb-bell-shaped cyst consisted of an upper relatively thick-walled part supplied by a branch

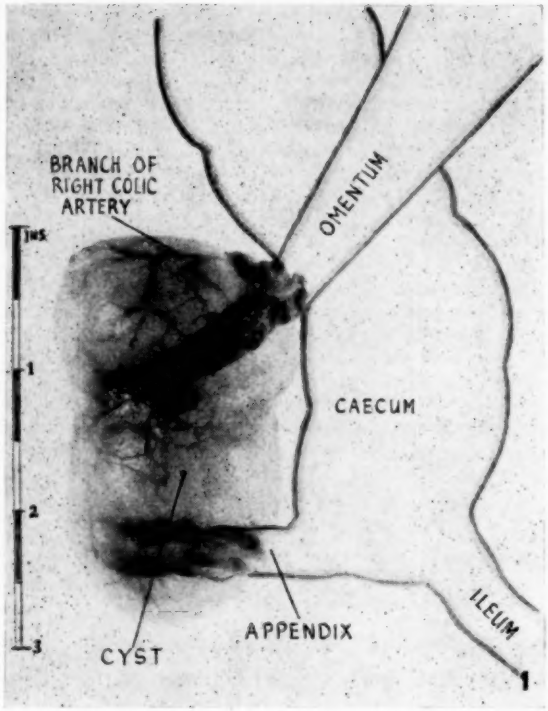


Fig. 1.

of the right colic artery. The waist and the lower pole were thinner walled and received blood supply from the omentum and the appendix (Fig. 1). The appendicular mesentery was completely separate from the cyst, and the cyst covered by peritoneum continuous with that of the caecum, was applied to the posterior abdominal wall. Histological preparations were made from the upper thick-walled part and the part to which the appendicular tip was adherent. The upper pole consisted of a well-defined muscular coat of smooth muscle arranged in two definite layers. No mucous membrane could be identified. The section through the appendix revealed that the muscular coat consisted of a single layer of smooth muscle, which blended with the outer longitudinal muscle of the appendix. The mucous lining of the cyst was thinned out and a layer of flattened epithelial cells was visible in certain parts. Although lymphoid tissue was present in abundance in the appendix, none was present in the cyst wall itself.

COMMENT

Ladd and Gross[1,2] grouped a variety of embryological anomalies associated with the alimentary tract under the heading 'Duplications of the alimentary tract'. These anomalies occur as diverticula, cysts and duplications and have a common developmental origin, that of sequestration of part of the developing alimentary canal in the 6 - 10 week embryo. The anomalies simulate the bowel structurally; they contain smooth muscle layers and mucous membrane resembling the mucous membrane of the bowel though not necessarily of the contiguous parts. The anomalous structure is usually partially or wholly adherent to the contiguous gut, and histologically it may be noted that the muscularis intervening is a common structure to both 'duplication' and contiguous bowel. The mucous membrane may be well defined, or flattened or partially or wholly absent as a result of enzymic or pressure necrosis. These anomalies are usually situated along the mesenteric side of the bowel from which they derive.

Duplications of the alimentary canal occur most commonly in the ileocaecal region.[1-5] Manifestations of 'duplication' anomalies of the appendix are relatively rare, occurring as multiple diverticula[6] or as true duplications.[7] A case of a giant cyst of the appendix has been described,[8] but this appears to have resulted from a possible inflammatory process and the formation of a mucocele. I have been unable to find any descriptions of true enterogenous cysts of appendicular origin in the literature.

In the present case the cyst presented most of the features of an enterogenous cyst on macroscopical and histological examination. Owing to tenseness caused by the accumulation of a large quantity of mucous fluid, the mucous membrane has survived only in parts of the cyst. The situation of the cyst relative to the caecum was unusual, and the cyst was found to be completely separable from that part of the bowel. It was, however, firmly attached to the tip of the appendix and adherent to it on its mesenteric aspect, although the meso-appendix was not involved. On the other hand, the absence of lymphoid tissue in the cyst wall, and the fact that the blood supply was derived mainly from the right colic artery, with a secondary supply through the adherent greater omentum and appendix, preclude the presumption that the cyst is derived from the appendix.

SUMMARY AND CONCLUSION

An enterogenous cyst in the ileocaecal region of a 4-month-old boy is described. This cyst may have resulted from a developmental fault in the developing appendix. The cyst was discovered incidentally when the child presented with gastro-enteritis.

REFERENCES

1. Ladd, W. E. and Gross, R. E. (1941): *Abdominal Surgery of Infancy and Childhood*, p. 83. Philadelphia and London: W. B. Saunders.
2. Gross, R. E. (1953): *Surgery of Infancy and Childhood* (1953), p. 221. Philadelphia and London: W. B. Saunders.
3. Evans, A. (1929): Brit. J. Surg., **17**, 34.
4. Hughes-Jones, W. E. A. (1934): Ibid., **22**, 134.
5. Dohn, K. and Povlsen, O. (1951): Acta chir. scand., **102**, 21.
6. Wilson, R. R. (1950): Brit. J. Surg., **38**, 65.
7. Menten, M. L. and Denny, H. E. (1945): Arch. Path., **40**, 345.
8. Lauret, J. (1948): Presse méd., **56**, 732.

BURSARIES AND STUDY GRANTS OFFERED BY THE ITALIAN GOVERNMENT

The Italian Government has decided to award a number of Bursaries to suitably qualified South African citizens who wish to complete a course or continue their studies in Italy, for the academic year 1960 - 61, beginning on 1 November 1960.

The duration of the Bursary is 8 or 10 months and the amount granted is It. Lire 60,000 (approximately £34 0s. 0d.) per month. If the Bursars wish to spend a few months in Italy before the beginning of the academic year, for the purpose of attending courses in the Italian language, this monthly grant will also be given them for that period.

The Bursaries of 10 months' duration will be assigned to South African students who intend to complete a full course leading to a degree or diploma at an Italian university and, subject to satisfactory reports on progress, will be renewed from year to year; or to university teachers and qualified artists who wish to continue their studies at universities, institutes, academies of fine arts or of music. The Bursaries, which expire on 31 August 1961, may also be renewed for the following year, subject to the above-mentioned condition.

Bursaries of 8 months (12 November 1960 - 30 June 1961) will be granted to students or scholars who intend to spend only 1 academic year in Italy and who wish to return to South Africa at the end of the Italian academic year.

The Italian shipping company Lloyd Triestino will allow successful applicants a 75% discount on the return fare.

In selecting candidates, preference will be given to applicants specializing in scientific research and study, or in experimental and technical work relating to mathematics, chemistry, civil engineering, architecture, industrial engineering, biology, medicine and pharmacology, agriculture and zoology, geography, geology, mineralogy, etc. But candidates who are qualified in other disciplines may also apply.

Applications (4 copies) should reach the Italian Consulates in Cape Town for the Cape Province and South West Africa,

in Johannesburg for the Transvaal and Orange Free State, and in Durban for Natal, not later than *15 April 1960*, together with the following documents:

1. One photograph, passport size;
2. Curriculum of past studies, in duplicate;
3. The original or 1 certified copy of latest school or university certificate;
4. A full description of the studies which the candidate intends to pursue in Italy;
5. Two testimonials by university or high school authorities or teachers;
6. A letter from a competent teacher of Italian, stating the candidate's knowledge of the Italian language;
7. Medical certificate; and
8. (For minors only): Consent of father or guardian to the acceptance of the Bursary.

The applications received by the above-mentioned Consulates will be examined by a specially appointed Selection Committee, which will meet at the Italian Embassy.

In addition, the Italian Government proposes to award a number of bursaries for periods of from 1 - 3 months to South African citizens who wish to pursue a short course of study in Italy during 1960.

Such bursaries will be awarded to those who have made a serious study of Italian language and culture at a South African university, or at classes run by the Dante Alighieri Society or an equivalent institution, and who wish to improve materially their knowledge at an Italian university, or at the University for Foreigners at Perugia, or one of the courses organized by the Societa Dante Alighieri in Italy. Teachers of Italian will receive special consideration.

A certain number of grants will be made to those who wish to take courses in music during the summer at the Conservatorium 'Benedetto Marcello' at Venice, or colleges of music of equivalent standard, or who wish to take courses arranged for foreign teachers at Perugia at the International Centre 'Luigi Severini'.

The number of grants—each of the value of 60,000 Lire (£34) a month—will be at least 4.

Applications for these grants must be sent in, in 4 copies, by 10 April 1960, to the Italian Consulate in Cape Town for the Cape Province and South West Africa, to the Italian Consulate in Johannesburg for the Transvaal and the Orange Free State, and to the Italian Vice-Consulate in Durban for Natal. They should include an application on the required form and also (1) a full account of previous study, (2) a certificate of competence in Italian, (3) two copies of the programme of study which the student intends to pursue; this should state (*a*) the particular course the applicant intends to follow, (*b*) the centre at which he intends to study, and (*c*) the date he intends starting work.

There is no need to enclose certificates of examinations passed or other such documents, except in the case of those who intend to study music; such students must enclose a certificate from the head of a school of music showing the standard which they have reached.

It should be remembered that the payment of such grants will only be authorized after the applicant has accepted his grant by letter. Since this takes about 2 months to arrange finally, those who receive such grants should not proceed to Italy before then, unless they have enough money of their own to live on until the matter is finally approved.

A limited number of application forms are available from the Registrar of the College of Physicians, Surgeons and Gynaecologists of South Africa, P.O. Box 120, Cape Town.

UNIVERSITY OF CAPE TOWN AND ASSOCIATION OF SURGEONS JOINT LECTURES

A series of lectures will be given in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, during the course of the first University term, and will be held in conjunction with the Association of Surgeons of South Africa (M.A.S.A.).

These lectures will be held on Wednesday afternoons at 5.30 p.m. Many will be illustrated with lantern slides and although they are chiefly of surgical interest all members of the Association are welcome.

PROGRAMME

24 February	Radiological features of the lower oesophagus	Mr. G. S. Muller Botha.
2 March	Subject to be arranged	Mr. S. Wass, of Guy's Hospital, London.
9 March	Selective gastric surgery with special reference to the augmented histamine test	Dr. I. N. Marks.
16 March	Blood transfusion	Dr. M. C. Botha
23 March	Subject to be arranged	Prof. I. Boerema, of Amsterdam.
30 March	Some thoughts on genital cancer	Prof. J. T. Louw.
6 April	Surgery of the common bile duct	Mr. J. E. Gasson.
13 April	Surgical convalescence	Mr. R. D. H. Baigrie.
20 April	The aortic valve with special reference to the surgical correction of aortic insufficiency	Dr. R. de Villiers.
4 May	Porphyria	Prof. L. Eales.
11 May	Tuberculosis of bone	Mr. T. L. Sarkin.
18 May	(*a*) Radiobiology	Dr. G. R. H. Sealy.
	(*b*) Principles of radiotherapeutic techniques	Dr. M. B. Bennett.
25 May	Breast cancer—aspects of local recurrence	Mr. P. Helman.
1 June	Report of overseas visit	Mr. J. Heselson.
8 June	Surgery in Russia	Dr. C. N. Barnard.

IN DIE VERBYGAAN : PASSING EVENTS

College of General Practitioners, Witwatersrand Faculty. The Executive Committee have arranged a series of lectures to be given by well-known members of the profession during 1960. The first lecture will be given by Prof. Guy Elliott, Head of the Department of Medicine, University of the Witwatersrand, on 'Modern concepts of the management of respiratory emergencies'. This lecture will take place on Thursday 25 February at 8.15 p.m. in the Lecture Theatre, South African Blood Transfusion House, Klein Street, Hospital Hill, Johannesburg. Visitors will be welcome, but members are asked to let the Secretary know beforehand if they intend to bring visitors. (Dr. L. Levy, 11 Millson House, Twist Street, Hillbrow, telephones 41-8787, 44-1938.)

* * *

Dr. Raphael Schapera, M.B., Ch.B. (Cape Town), D.C.P. (Rand), pathologist, has commenced practice at 503 Medical Centre, Heerengracht, Cape Town. Telephones: Rooms 3-6902, residence 71-4349, if no reply 3-1256. The telephone number of Dr. Schapera's consulting rooms does not appear in the current Telephone Directory.

* * *

Dr. Raphael Schapera, M.B., Ch.B. (Kaapstad), D.C.P. (Rand), patoloog, het begin praktiseer te Mediese Sentrum 503, Heerengracht, Kaapstad. Telefone: Spreekkamer 3-6902, woning 71-4349, indien geen antwoord 3-1256. Die telefoonnommer van dr. Schapera se spreekkamer verskyn nie in die jongste Telefoongids nie.

* * *

Dr. Max L. Bloom, M.B., B.Ch. (Rand), D.Obst.R.C.O.G., M.R.C.O.G., formerly Tutorial Assistant Obstetrician and Gynaecologist, University of the Witwatersrand Medical School and General Hospital, Johannesburg, has joined Drs. S. Joel Cohen

and M. Cecil Michelow in practice as a specialist obstetrician and gynaecologist at 208 Medical Centre, Johannesburg, and 106 Medical Centre, Krugersdorp. Telephones: 23-7124 and 23-5555, residence 40-1746 (not in current Telephone Directory), emergency 22-4191.

Dr. Max L. Bloom, M.B., B.Ch. (Rand), D.Obst.R.C.O.G., M.R.C.O.G., voorheen Doserende Assistent-Verloskundige en Ginekoloog, Mediese Skool, Universiteit van die Witwatersrand, en Algemene Hospitaal, Johannesburg, het nou in vennootskap met drs. S. Joel Cohen en M. Cecil Michelow as spesialis-verloskundige en -ginekoloog begin praktiseer by Mediese Sentrum 208, Johannesburg, en Mediese Sentrum 106, Krugersdorp. Telefone: 23-7124 en 23-5555, woning 40-1746 (nie in jongste Telefoongids nie), noodoproepe 22-4191.

* * *

University of the Witwatersrand Students' Medical Council, 17th Annual Conference. This Conference will be held at the Medical School, Johannesburg on 16 - 20 May 1960 and the subject will be 'The kidney'. Details of the programme will be published later.

* * *

Dr. I. V. Rogoff, urologist, of Johannesburg, has joined Dr. W. F. de Villiers in urological practice at 1 Balmain, Belmont Terrace, Port Elizabeth. Telephone 22020.

* * *

Dr. H. C. de Wet, wat die laaste 3 jaar in Londen, Edinburgh en Leeds hom op die chirurgie toegelê het, het onlangs met sukses die eksamen vir die F.R.C.S. (Edin.) afgelê en keer eersdaags terug na Suid-Afrika.

* * *

Verbetering. Amptelike Aankondiging: Wysiging van Verordening 6 (*c*). Die eerste paragraaf bo-aan bladsy 120 van die uitgawe van die *Tydskrif* van 6 Februarie 1960 moet soos volg lui: ‚6 (*c*) Lede wat die Vereniging sonder onderbreking ten minste 40 jaar lank gedien het, sal Lewenslange Lede word. Die betrokke Tak moet die Hoofkantoor van die Vereniging van die nodige informasie voorsien.'

IN MEMORIAM

DENYS HENRI RENÉ VOLLET, *O.B.E., Légion d'Honneur, Croix de Guerre*, M.B., CH.B. (CAPE TOWN)

Dr. C. Weinberg, of Durban, writes:

Dr. D. H. R. Vollet died suddenly on 10 December 1959.

The career of this remarkable man should not pass unnoticed both by the profession and the public. He was descended from a line of French military doctors. His generation was the 7th in an unbroken line of medical practitioners. His great grandfather served in Napoleon's army and was with Marshall Ney at Waterloo.

Dr. Vollet

Denys' father was in the Basutoland Medical Service. When the First World War broke out, he proceeded, in 1914, to the defence of his fatherland, taking with him Denys who was only 16 years old, and who enlisted as a private in a French infantry regiment.

This youngster was an active combatant in many of the bloody battles of those far-off days, and at the end of the war had attained the rank of Acting Captain. To those who have knowledge of the requirements for promotion in the French Army, this was an exceptional feat.

At the close of the war, Denys decided to follow in the steps of his forefathers, and became a medical student at the University of Cape Town. When he qualified he joined the Basutoland Medical Service and ultimately became its Director.

When the Second World War broke out Denys was on leave in England. He joined the R.A.M.C. and again took part in many of the major battles of the war (this time as a doctor), both in Europe and in Africa.

At the end of the war, he returned to Basutoland, and later retired to the home he had built at Cowies' Hill near Durban. In addition to being the Director of Medical Services, he was also Director of the first branch of the Red Cross to be formed in Basutoland.

He was on the Executive Committee of the BCESL, and was a foundation member of the College of Physicians and Surgeons and Gynaecologists of South Africa.

The *O.B.E.* was awarded to him in 1952 'for his services to Basutoland and its people'.

Denys Vollet probably won more decorations and honours than any other doctor in South Africa. He was a shy, modest man and I doubt if any but his intimate friends knew of his achievements. He had a French father and an English mother and he combined in himself much that is great in these two nations.

He was a humanist. He hated injustice and loved people and the arts. He had a zest for living. His passing leaves a gap in the lives of many of us, who combine in offering our sympathy to his mother, his wife Phyllis, and his son Robert.

FARMASEUTIESE NUUS: PHARMACEUTICAL NEWS

MAYBAKER (S.A.) (PTY.) LTD.

Maybaker (S.A.) (Pty.) Ltd. announce that as from Monday 29 February 1960, the address of their Transvaal Depot will be 43 Juta Street, Johannesburg. Their former premises at 111 Anderson Street, Johannesburg, will be closed down as from 22 February and, until the re-opening on the following Monday at 43 Juta Street, the branch will be shut for the purposes of ordinary business.

The telephone numbers, also, have been changed and will be 835-5425/6 as from 29 February 1960. The Post Office Box number, however, will remain unaltered i.e. P.O. Box 3926, Johannesburg.

During the change-over period of one week, orders will be serviced from the Port Elizabeth head office, and to obviate any delays, customers should take steps to draw stocks from the depot well before Friday 19 February.

The object of moving to larger premises is in order to provide a better service for customers in the Transvaal.

SOUTH AFRICAN DRUGGISTS LTD.

An announcement released in Johannesburg on 25 January gives the names of 10 pharmacy students to whom bursaries have been awarded out of funds made available by South African Druggists Ltd. The bursaries are of £60 each for the year 1960 and applications will be entertained by the sponsors if any bursar requires an assistance grant because of personal financial circumstances.

The following bursaries have been awarded: Potchefstroom University: M. Oosthuysen, H. Visscher. Rhodes University: H. Marlow, D. Walker. Witwatersrand Technical College: R. Carklin, B. Esterhuizen. Cape Technical College: R. Stipp, M. Kahn. Natal Technical College: T. Sella-Rolando. Port Elizabeth Technical College: I. Conry.

BOEKE ONTVANG : BOOKS RECEIVED

A Synopsis of Anaesthesia. 4th edition. By J. Alfred Lee, M.R.C.S., L.R.C.P., M.M.S.A., F.F.A.R.C.S., D.A. Pp. vi + 616. 72 illustrations. 27s. 6d. + 1s. 3d. postage. Bristol: John Wright and Sons Ltd. 1959.

Automatic Ventilation of the Lungs. By William W. Mushin, M.A., M.B., B.S., F.F.A.R.C.S., F.F.A.R.A.C.S., L. Rendell-Baker, M.B., B.S., F.F.A.R.C.S. and Peter W. Thompson, B.A., M.B., B.Chir., F.F.A.R.C.S. Chapters on *Physics of Automatic Ventilators*, by W. W. Mapleson, Ph. D., A.Inst.P. Pp. xvi + 349. 191 figures. 47s. 6d. Oxford: Blackwell Scientific Publications. 1959.

The Year Book of Endocrinology 1958-59. Edited by Gilbert S. Gordan, M.D., Ph.D., F.A.C.P. Pp. 384. 87 figures. $7.50. Chicago: Year Book Publishers, Inc. 1959.

Recent Advances in Cardiology. 5th edition. By Terence East, M.A., D.M., F.R.C.P. and Curtis Bain, M.C., D.M., F.R.C.P. Pp. vii + 421. 133 figures. 45s. net. London: J. & A. Churchill Ltd. 1959.

Basic Physics in Radiology. By L. A. W. Kemp, B.Sc., Ph.D., F.Inst.P. and R. Oliver, M.Sc., F.Inst.P., A.M.I.E.E. Pp. xiii + 329. Illustrations. 35s. Oxford: Blackwell Scientific Publications. 1959.

The Child with a Handicap. A team approach to his care and guidance. Edited by Edgar E. Martmer, M.D. Pp. xxiv + 409. Illustrations. 82s. 6d. Oxford: Blackwell Scientific Publications. 1959.

An Introduction to Medical Genetics. 2nd edition. By J. A. Fraser Roberts, M.A., M.D., D.Sc., F.R.C.P. Pp. xii + 263. 107 figures. 35s. London, New York, Toronto: Oxford University Press. 1959.

Office Gynecology. 7th edition, revised and enlarged. By J. P. Greenhill, B.S., M.D., F.A.C.S., F.I.C.S. (Hon.). Pp. 572. 145 figures. $9.00. Chicago: Year Book Publishers, Inc. 1959.

Speech and Brain-mechanisms. By Wilder Penfield and Lamar Roberts. Pp. xiii + 286. Illustrations. $6.00. Princeton: Princeton University Press. 1959.

Dermatologie und Venerologie einschliesslich Berufskrankheiten, dermatologischer Kosmetik und Andrologie. In 5 Bänden (Einzelne Bände erschienen in zwei Teilen). Herausgegeben von Prof. Dr. Dr. h.c. W. Schönfeld. *Band III. Teil* 1. *Hautvernäderungen durch äussere Einwirkung—Allergische Krankheiten und toxische Schädigungen der Haut—Formenkreis des Ekzems*. Bearbeitet von zahlreichen namhaften Fachgelehrten. xvi + 695 Seiten. 232 zum Teil farbige Abbildungen. Ganzleinen DM 165.00. Subskriptionspreis DM 132.00. Der Bezug von Teil 1 verpflichtet zur Abnahme des gesamten Bandes III (Teil 2 erscheint im August 1959). 20% ermässigter Subskriptionspreis für das Gesamtwerk. Der Subskriptionspreis gilt bis zum Erscheinen des letzten Bandes. Jeder Band ist einzeln zum Ladenpreis käuflich. Stuttgart: Georg Thieme Verlag. 1959.

Clinical Dermatology for Students and Practitioners. By Harry M. Robinson, Jr., B.S., M.D. and Raymond C. V. Robinson, B.S., M.D., M.Sc. (Med.). Pp. xi + 242. 117 figures. $8.50. 68s. Baltimore: The Williams and Wilkins Company. London: Baillière, Tindall & Cox Ltd. 1959.

Principles and Practice of Obstetric Anaesthesia. By J. Selwyn Crawford, M.B., Ch.B., D.A. (England), F.F.A.R.C.S. Pp. 128. Illustrations. 20s. Oxford: Blackwell Scientific Publications. 1959.

Peripheral Vascular Diseases. An objective approach. By Travis Winsor, M.D., F.A.C.P. Pp. xviii + 845. 435 figures. £6 5s. Oxford: Blackwell Scientific Publications. Springfield: Charles C. Thomas. 1959.

The Kinetics of Cellular Proliferation. Edited by Frederick Stohlman, Jr., M.D. Pp. xiv + 456. Illustrations. $5.75. New York and London: Grune & Stratton, Inc. 1959.

Klinik und Therapie der Vergiftungen. 3., neubearbeitete und erweiterte Auflage. Von Priv.-Doz. Dr. S. Moeschlin. Pp. xii + 655. 92 Abbildungen. Ganzleinen DM 59.00. Stuttgart: Georg Thieme Verlag. 1959.

Die Phlebographie der unteren Extremität. Von Dr. R. May und Dr. R. Nissl. Pp. x + 197. 250 Abbildungen in 395 Einzeldarstellungen. Ganzleinen DM 75.00. Stuttgart: Georg Thieme Verlag. 1959.

Die Streifenatelektasen der Lunge. Von Priv.-Doz. Dr. Fr. Heuck. Pp. viii + 108. 64 Abbildungen, in 136 Einzeldarstellungen. Kartoniert DM 32.00. (Zwanglose Abhandlungen aus dem gebiet der normalen und pathologischen Anatomie, herausgegeben von Prof. Dr. W. Bargmann und Prof. Dr. W. Doerr, Heft 7. Subskriptionspreis für Bezieher der Reihe DM. 25.60). Stuttgart: Georg Thieme Verlag. 1959.

Klinik und Praxis der Urologie. Band I. Klinik, Indikation, Diagnostik, operative und instrumentelle Eingriffe, Nachbehandlung. In zwei Bänden. Von Prof. Dr. W. Staehler. Pp. xxiv + 892. 1034 teils mehrfarbige Abbildungen. Ganzleinen DM 240.00. Der Bezug von Band I verpflichtet zur Abnahme von Band II. (Band II: Etwa DM 240.00, erscheint im September 1959) Preis des Gesamtwerkes etwa DM 480.00. Stuttgart: Georg Thieme Verlag. 1959.

Clinical Scalar Electrocardiography. 4th edition. By B. S. Lipman, A.B., M.D., F.A.C.P. and E. Massie, A.B., M.D., F.A.C.P. Pp. 474. 339 figures. $8.00. Chicago: Year Book Publishers, Inc. 1959.

Radiation Biology of Vicia Faba *in Relation to the General Problem*. By. J. Read, B.Sc., Ph.D. Pp. xxii + 270. 74 figures. 45s. Oxford: Blackwell Scientific Publications. 1959.

A Pocket Medicine. 4th edition. By G. E. Beaumont, M.A., D.M. (Oxon.), F.R.C.P., D.P.H. (Lond.). Pp. viii + 212. 12s. 6d. London: J. & A. Churchill Ltd. 1959.

More Medical and Other Verses. By A. E. Roche. Pp. viii + 35. 5s. net. London: H. K. Lewis & Co. Ltd. 1959.

A Short Text-book of Surgery. 7th edition. By C. F. W. Illingworth, *C.B.E.*, M.D., Ch.M., F.R.C.S. (Ed.). Pp. viii + 547. 254 text-figures with 16 plates. 45s. net. London: J. & A. Churchill Ltd. 1959.

Hale-White's Materia Medica Pharmacology and Therapeutics. 31st edition. By A. H. Douthwaite, M.D., F.R.C.P. (Lond.). Pp. viii + 525. 25s. net. London: J. & A. Churchill Ltd. 1959.

Forensic Medicine. Observation and Interpretation. By A. K. Mant, M.D. (Lond.). Pp. vii + 262. 118 figures. 42s. net. London: Lloyd-Luke (Medical Books) Ltd. 1960.

International Pharmacopoeia. 1st edition. Supplement. Pp. xx + 224. 25s. Published also in French. Spanish edition in preparation. Geneva: World Health Organization. 1959.

Bulletin of the World Health Organization. Vol. 21, No. 1. Pp. 144. Illustrations. 10s. Geneva: World Health Organization. 1959.

The Clinical Syndrome of Diabetes Mellitus. By J. Lister, M.A., M.D., M.R.C.P. (Lond.). Pp. x + 234. 34 illustrations. 35s. net. London: H. K. Lewis & Co. Ltd. 1959.

Treatment of Cancer in Clinical Practice. Edited by P. B. Kunkler, M.A., M.D. (Cantab.), M.R.C.P., F.F.R. and A. J. H. Rains, M.S. (Lond.), F.R.C.S. Pp. xvi + 821. Illustrations. 100s. net + 5s. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1959.

Recent Progress in the Endocrinology of Reproduction. Proceedings of the Conference held in Syracuse, New York, June 9-12, 1958. Edited by C. W. Lloyd. Pp. xi + 532. Illustrations. $12.00. New York and London: Academic Press Inc. 1959.

The Megaloblastic Anemias. By Victor Herbert, M.D. Pp. vi + 162. Illustrations. $6.00. New York and London: Grune & Stratton, Inc. 1959.

The Fluids of Parenteral Body Cavities. By P. D. Hoeprich, M.D. and J. R. Ward, M.D. Pp. iv. + 98. Illustrations. $4.75. New York and London: Grune & Stratton. Inc. 1959.

Tumors of the Pancreas. By V. K. Frantz, M.D. Pp. 149. Illustrations. $1.50. Washington: Armed Forces Institute of Pathology. 1959.

Miscellaneous Notes (*Fourth Series*). By F. Parkes Weber, M.D., F.R.C.P., F.S.A. Pp. 32. 6s. net. London: H. K. Lewis & Co. Ltd. 1959.

Text-book of British Surgery. Vol. 4. Edited by H. Souttar, *C.B.E.*, D.M. (Oxon.), F.R.C.S. and J. C. Goligher, Ch.M. (Edin.), F.R.C.S. (Edin. and Eng.). Pp. viii + 699. 405 illustrations. 105s. net. London: William Heinemann Medical Books Ltd. 1959.

Lectures on the Scientific Basis of Medicine. Vol. 7, 1957-58. Pp. xii + 496. Illustrations. 45s. net. London: University of London, The Athlone Press. 1959.

The Child with Abdominal Pains. By J. Apley, M.D., F.R.C.P. Pp. vii + 86. 12s. 6d. Oxford: Blackwell Scientific Publications Ltd. 1959.

Strong and Elwyn's Human Neuroanatomy. 4th edition. By R. C. Truex. Pp. xiii + 511. Illustrated. 80s. London: Baillière, Tindall and Cox Ltd. 1959.

A Practice of Anaesthesia. By W. D. Wylie, M.B. (Cantab.), M.R.C.P., F.F.A.R.C.S. and H. C. Churchill-Davidson, M.A., M.D. (Cantab.), F.F.A.R.C.S. Pp. xv + 1056. 246 figures. 105s. net. London: Lloyd-Luke (Medical Books) Ltd. 1960.

Soil, Grass and Cancer. Health of animals and men is linked to the mineral balance of the soil. By A. Voisin. Pp. xvii + 302. Illustrations. 30s. net. London: Crosby Lockwood & Son Ltd. 1959.

Ons en Ons Voedsel. Deur J. G. Schoonens. Pp. vi + 122. Illustrasies. 11s. 6d. Kaapstad, Bloemfontein, Johannesburg: Nasionale Boekhandel Bpk. 1959.

Methods of Radiochemical Analysis. Report of a Joint WHO/FAO Expert Committee. Technical Report Series No. 173. Pp. 116. 5s. Also available in French and Spanish. Geneva: World Health Organization. 1959.

Blindness in West Africa. By F. C. Rodger, M.D., Ch.M., D.O.M.S. Pp. xiv + 262. 1 colour plate. 6 maps. 96 text figures. 70s. net. London: H. K. Lewis & Co. Ltd. 1959.

Klinik und Praxis der Urologie. Band II. Klinik, Indikation, Diagnostik, operative und instrumentelle Eingriffe, Nachbehandlung. In zwei Bänden. Von Prof. Dr. W. Staehler. Pp. xxii + 863. 641 teils mehrfarbige Abbildungen. Mit Sach- und Namensverzeichnis für Band I und II. Der Bezug von Band I (erschienen im Juli 1959) verpflichtet zur Abnahme von Band II. Ganzleinen DM 240.00. Preis des Gesamtwerkes DM 480.00. Stuttgart: Georg Thieme Verlag. 1959.

The Effect of Pharmacologic Agents on the Nervous System. Proceedings of the Association for Research in Nervous and Mental Disease. Vol. XXXVII. Edited by F. J. Braceland, M.D. Pp. xi + 488. 124 illustrations. 34 tables. 108s. net. London: Baillière, Tindall and Cox Ltd. 1959.

A Text-book of Human Embryology. By R. G. Harrison, M.A., D.M. Pp. xi + 244. 144 figures. 45s. Oxford: Blackwell Scientific Publications. 1959.

Peripheral Facial Palsy. Pathology and surgery. By K. Kettel, M.D. Pp. 341. 127 figures. 120s. Oxford: Blackwell Scientific Publications. 1959.

Coronary Heart Disease. By J. W. Gofman, M.D., Ph.D. Pp. xx + 353. 60s. Oxford: Blackwell Scientific Publications. 1959.

Aldosterone in Clinical and Experimental Medicine. By E. J. Ross, M.D., Ph.D., M.R.C.P. Pp. viii + 144. 22s. 6d. Oxford: Blackwell Scientific Publications. 1959.

BRIEWERUBRIEK: CORRESPONDENCE

SKF LABORATORIES AWARD FOR POSTGRADUATE CLINICAL STUDY IN SOUTH AFRICA

To the Editor: Applications for the 1960 award of this Fellowship may now be submitted and must reach the undersigned not later than 30 June 1960.

The detailed conditions governing the award of this Fellowship appear in the issue of *Medical Proceedings*, dated 30 January 1960 on p. 34. They may also be obtained from the undersigned.

H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.Af.,
Honorary Chairman: Selection Committee,
SKF Laboratories Award for Postgraduate Clinical Study in South Africa

P.O. Box 1010
Johannesburg
27 January 1960

STERILIZATION FOR SEXUAL OFFENCES

To the Editor: How right Mr. Joubert[1] is in his view regarding sexual offences. But how wrong he is about sympathectomy. High bilateral lumbar sympathectomy generally causes sterility but not impotence—so the arm may yet be lifted, but the blow will be muted!

Hugh Benjamin

3rd Floor, Clarendon Centre
4 Park Lane
Parktown, Johannesburg
3 February 1960

1. Correspondence (1960): S. Afr. Med. J., **34**, 100.

[Isn't it time to close this correspondence before somebody 'calls a spade a spade'?—*Editor.*]

PAIN IN STERNUM AND THORACIC VERTEBRAE

To the Editor: May I offer a suggestion to *Puzzled*[1] whose letter has just been brought to my notice.

Next time he comes across a patient complaining of pain in the sternum and mid-thoracic spine he should place the patient prone on a hard couch and apply direct manual pressure followed by a quick jerk to the offending spinous processes followed by a similar manœuvre to either side of the processes in the same area. Both he and the patient will be aware of a 'clunch' under his hand and the pain will disappear almost immediately.

I have drawn attention to the mechanism of this syndrome elsewhere.[2]

In my experience this syndrome is extremely common though it does not always present with pain in both situations. More commonly the complaint is of sternal or substernal pain accompanied by pain on swallowing. The patient states that the bolus 'seems to pass a lump' behind the middle of the sternum. By applying one index finger to the site of the sternal pain and the other to the spinous process slightly above mid-sternum, usually T4 or 5, the latter will be found to be tender and simultaneous pressure will reproduce the pain. Frequently a band of hyperaesthesia will be found to join the two points.

The simple manœuvre outlined above will, in my experience, be sufficient to correct the abnormality of the intervertebral joint complex concerned. Should it fail the application of the 'full nelson' procedure should be carried out.

With the patient standing erect and his fingers interlaced behind his occiput, the operator's arms are passed under his axillae and threaded through his flexed elbows, the fingers of the operator being interlaced and placed over those of the patient. The patient is instructed to bend forward slightly and pull his elbows forward. The operator then applies pressure selectively to the thoracic vertebrae in turn with his own sternum while flexing the patient's neck. This procedure will achieve a satisfactory result, both to operator and patient.

Philip H. Dalgleish, M.B., Ch.B.

P.O. Box 79
Hill Crest, Natal
2 February 1960

1. Correspondence (1960): S. Afr. Med. J., **34**, 20.
2. Dalgleish, P. H. (1959): Med. Proc., **5**, 523.

CONDEMNATION OF ANAESTHETIC SOLUTIONS FOR HAEMORRHOIDS

To the Editor: Are some surgeons still using the long-acting oily anaesthetic solution and pretending that they are doing a so-called painless haemorrhoid operation? I have a case now where the infective process has spread very widely. Will this patient end up with a resection of the rectum and a permanent colostomy?

Would these surgeons allow this substance to be used on themselves; if not, what right have they to use it on patients? It is used by these people purely for attracting patients and for personal gain. In my opinion this procedure falls under malpractice.

In a lighter vein—an elderly patient of 70 years whom I examined 5 years ago and whose piles were small and of no consequence, recently returned to consult me about her rectal neurosis. She informed me that she had had a haemorrhoid operation 2 years ago. When I asked her why she did not request me to do the operation, she replied that I used the knife and that the doctor who treated her for piles took them out with his hands, and that he only kept his patients in the nursing home for 4 days, whereas other doctors kept their patients in for nearly 3 weeks.

Stephen Eisenhammer

34 Moray House
Cor. Jeppe and Smal Streets
Johannesburg
3 February 1960